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# Severe *Escherichia coli* O111 septicaemia and polyserositis in hens at the start of lay

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Three very severe episodes of *Escherichia coli* infection in hens from the same farm, at the beginning of laying, are reported. They were characterized by no clinical signs, but sudden mortality, from 5 to 10%, with severe lesions of septicaemia and fibrinous polyserositis. A Gram-negative bacterium was consistently isolated in pure culture from tissues. Isolates were typed biochemically as *E. coli*, but they were lactose negative and non-motile. The serotyping tests typed the isolates as somatic group O111. The isolates were sensitive to enrofloxacin, amoxycil and colimycin, and partially sensitive to flumequine, all of which were used for therapy. The disease was reproduced experimentally in both specific pathogen free chickens and commercial layers by intramuscular inoculation of the *E. coli*, but only in some layers when inoculated by the oro-nasal route. The stress of the onset of lay seemed to be the most probable precipitating cause of the disease.

## Introduction

The association of *Escherichia coli* with certain pathological conditions of poultry dates from the end of the last century. Many reports on this subject have been published (Hjärrè & Wramby, 1945; Gross & Siegel, 1959; Sojka & Carnagham, 1961; Sojka, 1965; Emery *et al.*, 1992; Barnes & Lozano, 1994; Morris, 1994; Dhillon & Jack, 1996). Generally, *E. coli* affects poultry of all ages, although young birds are more sensitive. The infection is considered to be one of the leading causes of economic loss in the poultry industry.

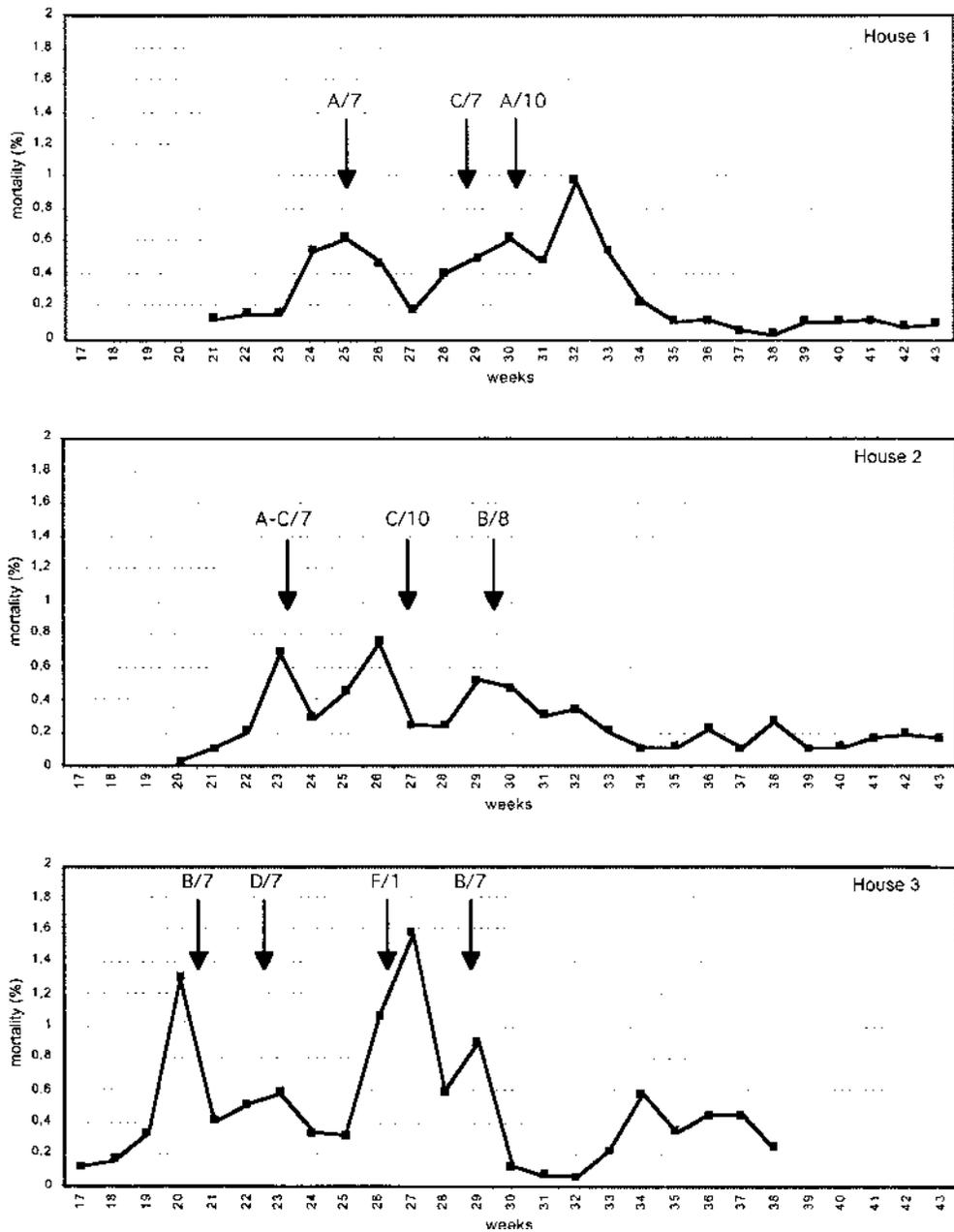
Different manifestations of *E. coli* infection have been described: septicaemia, enteritis, granulomas, omphalitis, sinusitis, airsacculitis, arthritis/synovitis, peritonitis, pericarditis, cellulitis, swollen head syndrome, etc. More frequently, *E. coli* disease occurs as a consequence of the adverse influence of factors such as ammonia, moisture, dust, hormones or infectious agents such as viruses and myco-

plasmas (Oyetunde *et al.*, 1978; Weinach *et al.*, 1984; Gross, 1990; Leitner & Heller, 1992). Sometimes *E. coli* is the primary cause of disease, particularly in young birds (Cheville & Arp, 1978), but also in adults (Dhillon & Jack, 1996). Many serotypes of *E. coli* have been isolated throughout the world (Sojka & Carnaghan, 1961; Glanz *et al.*, 1962; Hemsley *et al.*, 1967; Rosenberger & Chand, 1981; Cloud *et al.*, 1985).

Currently, 173 O, 74 K, 53 H and 17 F antigens are recognized (Barnes & Gross, 1997), but the most frequently occurring serotypes in poultry are O1, O2, O8, O35 and O78. Pathological conditions due to *E. coli* have been reproduced experimentally by different routes of inoculation, with mortality varying from 30 to 100% (Gross, 1957; Sojka & Carnaghan, 1961; McGruder, 1996), and many studies have been published on the *in vitro* and *in vivo* susceptibility and resistance to chemotherapeutics and antibiotics (Heller & Smith, 1973; Rosenberger & Chand, 1981; Cloud *et al.*, 1985; Aleson Sanz & Aleson Sanz, 1996).

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**Figure 1.** Curve of weekly mortality of brown egg layers in the three affected houses. Different treatments for each house are also shown, indicating day and type of drug: A, amoxicillin; B, enrofloxacin; C, colistin; D, difloxacin; F, flumequine.

The purpose of this paper is to report on three episodes of *E. coli* infection that occurred in succession in three flocks of laying hens on the same farm at the onset of lay, and to describe experimental reproduction of the condition.

## Materials and Methods

### Flocks and housing

The disease outbreak occurred in three adjacent houses, each stocked with approximately 75 000 brown egg layers of a different age. It first appeared in house 1 in December 1997 at onset of lay, i.e. 19 to 20 weeks of age. House 2 became affected 3 weeks later and house 3 was affected 5 months later, each at onset of lay (Figure 1). The birds in each house originated from different rearing houses: those of house 1 had been reared on the floor, and those of houses 2 and 3 were reared in cages, before being transferred to their respective laying cages at 16

weeks. All the birds were kept in modern houses in cages (five birds per cage) and with controlled ventilation.

Two older flocks, placed 20 and 37 weeks before the first affected flock but in adjacent houses, remained unaffected. Many groups of pullets from the brooder houses also supplying houses 2 and 3, and which were placed on a different farm belonging to the same company, remained unaffected.

### Culture and biochemical characterization

Liver, spleen and brain from approximately 30 dead birds in each house were cultured on MacConkey agar, blood-agar (Difco, Detroit, MI, USA) and Gassner VMC agar (Oxoid, Basingstoke, UK) media. Selective and enrichment techniques for detection of *Salmonella* were the following: liver and ovarian specimens were seeded in tetrathionate broth (Difco), incubated overnight at 42°C, plated onto XLT4 agar plates (Difco) and incubated at 37°C.

Metabolic profiles were compiled for each isolate using a miniaturized micro-organism differentiation system (Enterotube; Becton

Dickinson, San Jose, CA, USA) or API System (Bio Mérieux; Marcy L'Etoile, Lyon, France), designed for the identification of *Enterobacteriaceae*.

#### *Serological characterization of E. coli and serological tests*

Four cultures of *E. coli* isolates, obtained from houses 1 and 2, were serotyped by Biovac BP61 (Beaucouze Cedex, France). Five isolates from the episode in the third house were serotyped with a specific antiserum prepared in the Institute of Veterinary Microbiology, University of Milan. The inoculum used to produce this serum was an oil emulsion bacterin, prepared from a broth-culture containing  $4 \times 10^8$  organisms/ml, using a serotyped isolate from the first episode. The bacterin was injected intramuscularly in the dose of 1 ml on two occasions, 3 weeks apart, into six specific pathogen free (SPF) 7-week-old chickens.

The sera from field layers (1% of cohabiting birds in three houses) and from experimental birds (see later) were tested by rapid agglutination test, using as antigen a crude suspension of the O111 *E. coli* isolate in the smooth phase.

#### *Antimicrobial sensitivity and medication*

An 8 to 12 h broth culture was prepared for each of 15 isolates obtained from the three episodes (five from each one) and swabbed onto the surface of Müller-Hinton agar (Difco). Paper discs (Difco), impregnated with the standard concentration of antibiotic to be tested, were laid on the medium. The plates were incubated for 24 h at 37°C and inhibition zones were measured.

For medication, enrofloxacin (Baytril; Bayer, Leverkusen, Germany), amoxicillin (Supramox; Fatro, Bologna, Italy), colistin (Colistin sulphate; Sintofarm, Reggio Emilia, Italy), difloxacin (Dicural; FortDodge, Naarden, The Netherlands) or flumequine (Flumequine; Gellini, Aprilia, Italy) were administered one at a time to the three affected flocks in the drinking water at the recommended doses for 7- to 10-day periods. A minimum of three courses of treatment was given as indicated in Figure 1.

#### *Experimental infection*

Twelve 5-week-old White Leghorn SPF chickens and 21-week-old commercial layers (22 birds) of the same type as the field cases, at 75% egg production, were used in two different experiments. They were bacteriologically and serologically negative for *E. coli* O111. The SPF birds were kept in isolation in the same cage. The commercial birds were housed in three different cages in the same isolation room. The inoculum, a broth culture that was prepared from an isolate previously serotyped, was given either as an intramuscular (i.m.) injection in the leg, with  $5 \times 10^6$  colony forming units (CFU) of the *E. coli* isolate, or by the oro-nasal (o.n.) route with  $10^8$  CFU of the same *E. coli* isolate. In the second trial, a group of six birds was maintained as a contact control group. All the birds were examined daily for clinical signs, morbidity and mortality. Daily egg production was recorded in the layers. All dead birds were examined and lesions recorded. Surviving birds in all groups were bled 21 days after infection and the resulting sera were tested by rapid agglutination for antibodies to *E. coli* O111. At the end of the experiment, the surviving birds were killed and necropsied.

## Results

### *Signs and lesions in affected flocks*

The birds showed no clinical signs prior to death, and egg production was not significantly affected, except in house 1, where a decrease of 10% was recorded. The mortality, spread evenly over the three houses, ranged from 5 to 10% in a period of about 14 weeks (Figure 2), with a three-phasic trend in relation to the antibiotic treatments (Figure 1).

Approximately 2% of birds that died were necropsied twice a week, and splenomegaly, fibrinous pericarditis, perihepatitis, inflammation of the ovaries and lung congestion were clearly evident in almost all these birds.

### *Culture and biochemical characterization*

Isolates obtained at different times, during the septicaemic phases, from liver, spleen and brain of 90% of the examined birds proved to be pure cultures of a Gram-negative bacterium that was non-motile and did not ferment lactose. The commercial differentiation systems identified the organism as *E. coli*, and the biochemical activity of 22 isolates obtained from the three episodes was exactly the same. All samples were negative for *Salmonella* or other pathogens.

### *Serological characterization of E. coli and serological tests*

Two isolates of *E. coli* from each of the first two affected houses were serotyped as somatic group O111. Five isolates of the fourth episode, tested by us, were also somatic group O111.

Agglutination tests, carried out on the 1% of sera collected from birds in each of the three houses, demonstrated no detectable agglutinating antibodies to O111 antigen.

### *Antimicrobial sensitivity and medication*

In antibiotic sensitivity tests, carried out on 15 isolates from all three flocks, enrofloxacin, amoxicillin, ampicillin, apramycin, colistin and gentamicin were the most active *in vitro*; flumequine was active only at the beginning of use, i.e. with isolates recovered early in the course of infection. Lincomycin, kanamycin, tetracycline, chloramphenicol and nalidixic acid were totally inactive (Table 1).

A single course of medication using the antimicrobials at the recommended doses was not sufficient to control the disease. At least three treatment cycles of 7 to 10 days were necessary over a period of about 14 weeks before mortality returned to near normality (Figure 1).

### *Experimental reproduction of disease*

Pathological conditions resembling naturally occurring colisepticaemia were reproduced both in SPF chickens and in commercial layers following inoculation of *E. coli* by the i.m. route. Rapid onset of deep depression, lameness and diarrhoea were observed in both types of bird. At necropsy, pericarditis, perihepatitis and splenomegaly were seen. The same conditions were also reproduced in two of eight (20%) commercial layers, but not in younger SPF chickens, by administering  $10^8$  CFU of the organism by the o.n. route. Egg production

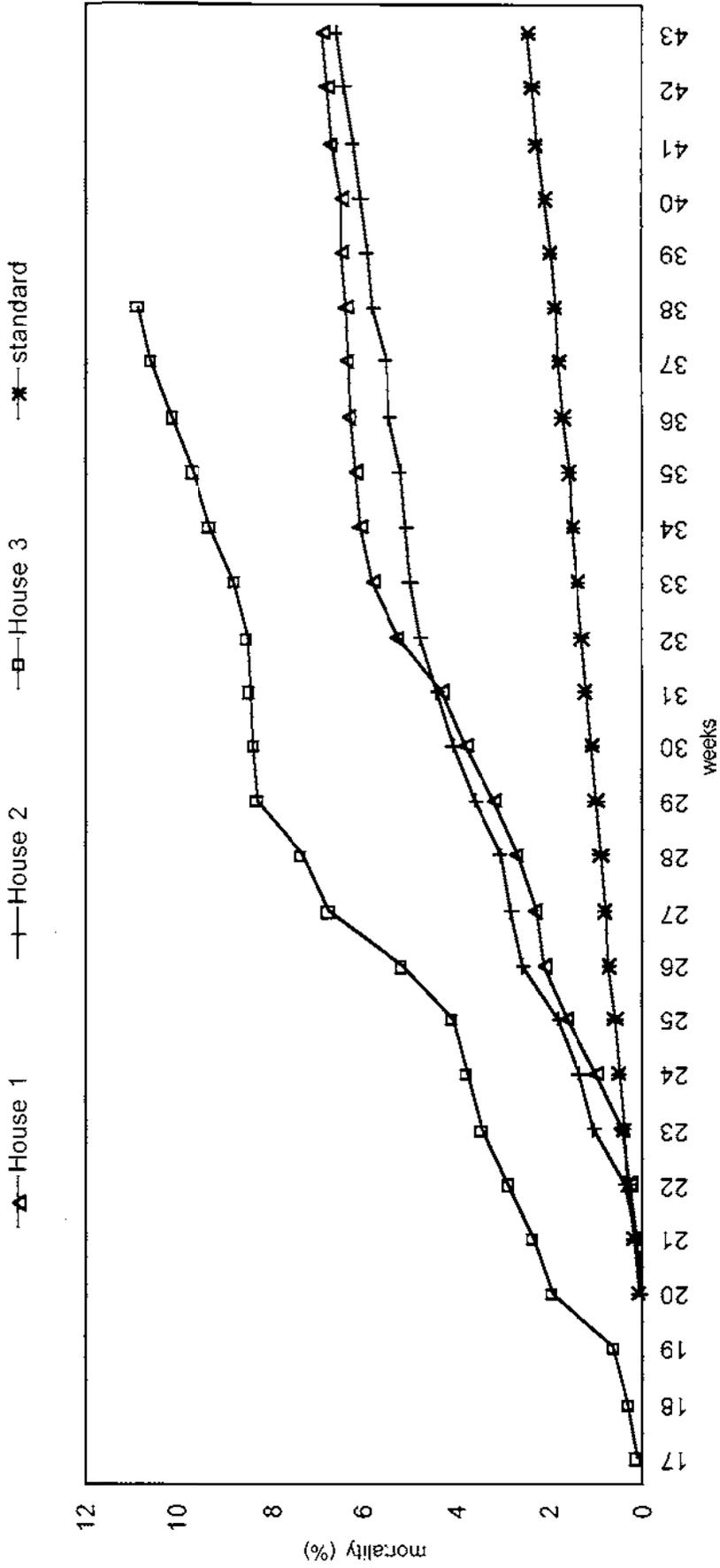


Figure 2. Progressive mortality in the three affected houses of brown egg layers compared with standard mortality.

**Table 1.** Results of antibiotic sensitivity tests on 15 isolates from three episodes of *E. coli* septicaemia

Antibiotic	Concentration (µg)	Isolate number <sup>a</sup>														
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Enrofloxacin	5	+ <sup>b</sup>	+	+	+	+	+	+	+	+	+	+	+	+	+	+
Amoxycillin	10	+	+	+	+	+	- <sup>c</sup>	-	+	-	+	+	+	+	+	+
Colistin	10	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
Flumequine	30	+	+	+	+	+	-	+	+	-	-	-	-	-	-	-
Apramycin	15	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
Gentamycin	10	+	+	+	+	+	+	+	-	+	+	+	+	+	+	+
Ampicillin	10	+	+	+	+	+	-	+	+	+	+	+	+	+	+	+
Tetracycline	30	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Lincomycin	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Chloramphenicol	30	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Nalidixic acid	30	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Kanamycin	30	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

<sup>a</sup> Isolates recovered at different times (between December 1997 and August 1998) from three episodes.

<sup>b</sup> Sensitive.

<sup>c</sup> Resistant.

ceased in hens infected by i.m. injection, but remained at normal values in the o.n. group (Table 2). The contact birds remained normal throughout the experiment. Seroconversion was demonstrated in all surviving birds infected by the i.m. route, and only in one of the birds infected by the o.n. route.

**Discussion**

The serological characterization of the *E. coli* isolates from the three affected houses showed that they belonged to the uncommon serotype O111. The isolation of such a serotype was apparently reported only once before in poultry (Cloud *et al.*, 1985), although more frequently in humans and calves (Griffin & Tauxe, 1991). The disease was controlled by using enrofloxacin, amoxycillin and colistin, although at least three cycles of treatment were necessary.

Pathological conditions resembling naturally occurring colisepticaemia were readily reproduced

in both SPF chickens and commercial layers by i.m. inoculation of the organism, illustrating its ability to invade, but o.n. inoculation caused disease in only two of eight 21-week-old layers and in none of the SPF birds. The layers, building up towards peak production, may have been more susceptible due to the stress imposed by the stage of lay. All the experimentally infected birds that recovered from the disease showed seroconversion using the rapid agglutination test.

As long as the intestinal mucosal barrier is intact, the normal microflora of birds is likely to inhibit the translocation of pathogenic *E. coli* from the intestine to the bloodstream and organs. When these barriers are damaged, possibly by the stress of coming into lay, pathogenic bacteria may invade and cause septicaemia. Stressed chickens that do not develop acute disease may recover and produce antibodies (Leitner & Heller, 1992). In our field cases, no detectable agglutination antibodies were found in a sample of 2% of cohabiting birds,

**Table 2.** Results of experimental infection of SPF chickens and commercial layers with *E. coli* serotype O111

Type of chicken	Age (weeks)	Number	Inoculation route	Dose of <i>E. coli</i> (CFU)	Number of birds			Egg production (%)	
					Morbidity	Mortality	Sero-conversion	First week	Second week
SPF	5	8	i.m.	5 × 10 <sup>6</sup>	8/8	5/8	3/3	-	-
	5	4	o.n.	10 <sup>8</sup>	0/4	0/4	0/4	-	-
Brown egg layer	20	8	i.m.	5 × 10 <sup>6</sup>	8/8	1/8	7/7	12	0
	20	8	o.n.	10 <sup>8</sup>	2/8	1/8	1/7	86	82
	20	6	Contact	-	0/6	0/6	0/6	80	78

although only one bird that recovered from disease after experimental o.n. infection and all the birds that recovered after i.m. infection produced antibody.

The different responses of the birds may be related to their MHC haplotype or to a particular individual resistance to the stress of laying, which prevents the intestinal pathogen from translocating to the blood stream. In either case, it is also likely that the bacteria entering the submucosa would induce secretory immunoglobulin A antibodies in surviving birds, and provide local immunity without inducing a systemic reaction. This might affect the outcome of infection, i.e. death or survival without symptoms. However, this is not easy to demonstrate experimentally.

In conclusion, although the pathogenesis of *E. coli* is poorly understood, there is a general agreement that stress and a resulting bacteraemia are essential factors for the development of clinical disease and mortality.

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## RÉSUMÉ

### Septicémie sévère à *Escherichia coli* 0111 et polysérosite chez des pondeuses en début de ponte

Trois épisodes très sévères d'infection par *Escherichia coli* chez des pondeuses en début de ponte appartenant au même élevage ont été décrits. La caractérisation a été l'absence de signes cliniques, mais une mortalité subite de 5% à 10%, et des lésions sévères de septicémie et de polysérosite fibrineuse.

Une bactérie gram-négatif a été régulièrement isolée en culture pure à partir des tissus. Les isolats ont été typés par biochimie comme *E. coli*, mais ils étaient lactose négatif et non motiles. Les tests de sérotypage ont permis de classer les isolats dans le groupe somatique 0111.

Les isolats se sont avérés sensibles à l'enrofloxacin, l'amoxicilline, la colimycine et partiellement à la fluméquine; tous ces antibiotiques ont été utilisés en thérapie.

La maladie a été reproduite expérimentalement chez des poules EOPS et chez des pondeuses commerciales par inoculation intramusculaire d'*E. coli*, mais seulement chez quelques pondeuses après administration oro-nasale.

Le stress du début de la ponte a semblé être la cause la plus probable de la maladie.

## ZUSAMMENFASSUNG

### Schwere *Escherichia coli* 0111 Septikämie und Polyserositis bei Hennen am Anfang der Legetätigkeit

Drei sehr schwere Vorfälle einer *Escherichia coli*-Infektion bei Hennen von derselben Farm zu Beginn ihrer Legetätigkeit werden beschrieben. Sie waren durch eine plötzliche Mortalität von 5 bis 10% mit schweren Veränderungen von Septikämie und fibrinöser Polyserositis, aber ohne klinische Symptome gekennzeichnet.

Ein gramnegatives Bakterium wurde durchweg in Reinkultur aus Geweben isoliert. Die Isolate wurden biochemisch als *E. coli* typisiert, sie waren aber Laktose-negativ und nicht beweglich. Die Serotypisierung ergab die Zugehörigkeit der Isolate zum Serovar 0111.

Die Isolate waren empfindlich gegen Enrofloxacin, Amoxicillin, Colimycin und partiell gegen Flumequin, die alle für die Therapie verwendet wurden.

Die Krankheit wurde sowohl bei SPF-Hühnern als auch bei kommerziellen Legehühnern durch intramuskuläre Inokulation des *E. coli* experimentell reproduziert, aber nur bei einigen Legehühnern, wenn sie oronasal inokuliert wurden.

Der Stress des Legebeginns schien die wahrscheinlichste auslösende Ursache der Krankheit zu sein.

#### RESUMEN

##### **Septicemia y poliserositis severa causada por *Escherichia coli* O111 en gallinas al inicio de la puesta**

Se presentan tres casos de infección severa por *Escherichia coli* en gallinas, pertenecientes a una misma granja, al inicio de la puesta. En

ninguno de los tres casos se observaron síntomas clínicos, únicamente una súbita mortalidad, del 5 al 100%, acompañada de lesiones severas de septicemia y poliserositis fibrinosa. Se aisló una bacteria gram negativa de forma constante y en cultivo puro a partir de tejidos. Las cepas aisladas fueron tipificadas bioquímicamente como *E. coli*, aunque eran lactosa negativas y no móviles. Mediante técnicas de serotipificación se clasificaron las cepas como grupo somático O111. Las cepas aisladas resultaron sensibles a la enrofloxacina, amoxicilina, colimicina y parcialmente a la flumequina, las cuales se usaron como tratamiento. El proceso patológico fue reproducido experimentalmente en pollos SPF y en gallinas comerciales mediante inoculación intramuscular de *E. coli*, pero solamente en algunas gallinas cuando éstas fueron inoculadas por vía oronasal. El estrés del inicio de la puesta podría ser la causa que probablemente precipitó el proceso patológico.