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Treatment of a field case of avian intestinal spirochaetosis caused by *Brachyspira pilosicoli* with tiamulin

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**Treatment of a field case of avian intestinal spirochaetosis caused by
Brachyspira pilosicoli with tiamulin**

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**Treatment of a field case of avian intestinal spirochaetosis caused by
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Abstract

There has been much confusion over the significance of spirochaetes found in the caeca of laying hens and the impact they may have on egg production. In recent years, the situation has been made clearer and the presence of such species as *Brachyspira pilosicoli* have been shown to cause a mild, chronic disease in both layers and breeders and reduce egg production by reportedly five percent. In the United Kingdom, a multi-age caged laying site with three separate flocks of approximately 12,000 birds each, was chronically infected with *B. pilosicoli* but displayed few clinical signs except for a noticeable reduction in egg production and an increased mortality. The flocks were treated for three days in the drinking water with tiamulin at 12.5mg/kg bodyweight and a steady improvement in performance was recorded. The production results were compared with a flock that had been untreated with tiamulin previously, as a control, and one that had been treated at 25 and 45 weeks of age. A 9.8% improvement in egg production/hen housed upto 72 weeks of age and 9.7% in total egg weight was recorded, as well as an 8.6% reduction in actual hen mortality, in the tiamulin-treated flock in comparison with the untreated control. After taking into account the difference in breeds used, there was only a 6% reduction in egg production but an 8.84% increase in mortality in the untreated

flock compared with the individual breed's standard production data. The cost of the disease was estimated at £14 million in the UK based on a national laying flock of 30 million or 1.5% of production. Faecal examination for potentially pathogenic spirochaetes should be a part of the differential diagnosis of under-performing laying flocks.

For Peer Review Only

Introduction

Avian intestinal spirochaetosis is defined in general terms (Swayne, 2003), as a spirochaete colonisation, predominately of the caecum and/or rectum of poultry. However, this is to be distinguished from the clinical disease, which is described in pullets, layers and breeders as causing soft to watery, brown to grey diarrhoea, pasty vents and is often associated with a delayed onset of lay in pullets, reduced egg production and body weight in layers and poor chick quality from infected breeders (Smit *et al*, 1998). The severity of the condition depends on the pathogenicity of the spirochaetes and their relative destruction of the surface epithelium of the caecum and degree of typhlitis caused. It is a chronic infection, with colonisation and excretion of the organisms for several months (Dwars *et al*, 1990; Hampson and McLaren, 1999)

Spirochaetes were commonly found in the caecum of layers and breeders (Dwars *et al*, 1989; Stephens and Hampson, 1999) but were not always associated with clinical disease. Initially, this led to confusion over the significance of spirochaete infections in chickens, until the 1990's, when the different spirochaetes found in poultry were better classified genetically and phenotypically (McLaren *et al*, 1997; Phillips *et al*, 2005).

Stephens and Hampson (2002a) showed that *Brachyspira pilosicoli* was potentially pathogenic in breeder layers, causing a delayed onset of lay and a significant reduction in egg production, in comparison with *B. innocens*, which was shown to be non-pathogenic. Hampson and McLaren (1999) found that an isolate of *B. intermedia* also caused a significant reduction in egg production and also mean egg weight.

Brachyspira alvinipulli has also been shown to be mildly pathogenic in the United States (Swayne *et al*, 1992; Swayne, 2003).

The association of spirochaetal infections in laying flocks with enteric disease in Holland was reported by Dwars *et al* (1989). In a study of 179 flocks, spirochaetes could be demonstrated in 27.6% of flocks with reported enteric disorders but only 4.4% in flocks with no enteric signs. In Australia (Stephens and Hampson, 1999), the prevalence of spirochaetes in 50 flocks tested appeared to be much higher; with 42.9% of breeder flocks and 68.2% of layer flocks infected using a different methodology. Isolates from a subset of 16 flocks were investigated to a species level and *B. pilosicoli* and *B. intermedia* were found in 8 flocks (50%) *B. pilosicoli* in 6 flocks (37.5%) and *B. intermedia* in four flocks (25%) either alone or in mixed infections. Bano *et al* (2005) reported on a survey in northeastern Italy involving 29 layer farms. Spirochaetes were found in 72.4% of the layer farms and 71.1% of the sheds. The pathogenic spirochaetes, *B. pilosicoli* and *B. intermedia* were found in 31% of the sheds in an approximate 1:2 ratio.

The susceptibility of avian spirochaetes to a variety of antimicrobials was reported by Hampson and Stephens (2002) using an agar dilution method. Tiamulin was shown to be the most active with 15/19 (79%) isolates having a minimum inhibitory concentration of less than 0.1µg/ml and the remainder were between 0.1-1.0µg/ml. Other anti-spirochaetal antimicrobials were also shown to be active such as metronidazole, lincomycin and to a lesser extent, tylosin.

Stephens and Hampson (2002b) carried out an artificial challenge study with *B. pilosicoli* in broiler breeders and administered tiamulin at 25mg/kg bodyweight/day by

crop tube for 5 consecutive days. Birds that had been infected and treated stopped shedding *B. pilosicoli*, whereas the untreated infected birds continued to shed until termination of the study, four weeks later.

It is the purpose of this report, to describe the use of tiamulin in the field in flocks which had been chronically infected with *B. pilosicoli* and their response to treatment.

Clinical case

A commercial laying farm, with three caged flocks of approximately 12,000 birds each, was reported to be showing reduced egg production and poor performance over an 18 month period. Each flock was subjected to serological examination at 20 weeks of age to monitor the efficiency of the vaccination programme during rearing to act as a baseline for any subsequent investigations. Generally these point-of-lay results indicated good to very good levels of protection against the common viral infections. The subsequent production problems were investigated serologically using both haemagglutination inhibition (HI) and enzyme-linked immunosorbent assay (ELISA) tests, looking for challenges from infectious bronchitis (IB) virus (M41) and variants (793B, D274 and D1466), avian rhinotracheitis (ART) virus, *Mycoplasma gallisepticum* and *M. synoviae*. There was evidence of rising titres from intermittent challenges by IB variants, mainly 793B and D274, in common with the situation on other units belonging to the company but these did not appear to correlate with the production problems on this site as there were no clinical signs noted. All flocks had been vaccinated at least twice against the IB variants found. *Mycoplasma synoviae*, which is commonly reported (79%) in the UK

laying flocks (Hagen *et al*, 2004), was also present in these flocks. There was no response to treatment with chlortetracycline administered via the feed at 600ppm on regular 7-8 week intervals to control any mycoplasmal infections and related conditions. The level of soft faeces was higher than normal but not considered widespread. Faecal samples were collected in each shed from 12 cages, each containing five hens, by placing cling-film covered cards under the cages. Aliquots of faeces, including any abnormal droppings were then transferred into six, 5ml containers, filled to capacity to exclude air. These samples were sent for examination at the Veterinary Laboratories Agency (VLA) in Sutton Bonington, specifically for *Brachyspira* spp examination, which is not routinely carried out on poultry faeces in the UK. Spirochaetes were observed weakly fluorescing, following fluorescent antibody staining (FAT) using a *B. hyodysenteriae*-specific antibody and positive samples were submitted to culture. Spirochaetes were subsequently cultured and shown to cause a weak haemolysis on trypticase soy agar plates with defibrinated sheep blood with antimicrobials, spectinomycin, colistin and vancomycin to prevent overgrowth by other bacteria. Cultures were sent to VLA Winchester for definitive identification by biochemical tests. The presence of *B. pilosicoli* was confirmed by biochemical differentiation and found to be indole negative but hippurate positive. Serum agglutination tests with *B. hyodysenteriae*-specific serum were negative. Initially, only one shed was shown to be positive, but on repeated sampling all three sheds yielded *B. pilosicoli*. Polymerase chain reaction (PCR) tests for *Brachyspira* spp were not available in the VLA in England and minimum inhibitory concentration (MIC) testing of isolates, especially from poultry, was not routinely carried out.

Several post-mortem examinations were performed over the problem period, especially when mortality rates were much higher than usual. The vast majority of deaths were associated with peritonitis caused by *Escherichia coli*.

Following consultation, it was decided to treat the oldest flock with tiamulin at 12.5mg/kg bodyweight for 3 days via the drinking water. The daily dose in pigs for *B. hyodysenteriae*, a related organism, which causes swine dysentery, was 8.8 mg/kg bodyweight for 3-5 days. It was reported that the birds showed consistent and steady improvements in faecal consistency, appetite, body weight, egg production and mortality. It was decided to medicate the two remaining younger flocks one was 45 weeks of age and the other was 25 weeks of age. Additional tiamulin medication was applied at 60 weeks and 45 weeks of age to each flock respectively, as a precaution against possible recurrence, following rigorous insect-control measures, as it was thought likely that flies might maintain the spread of the infection. Unfortunately, no further faeces samples were taken following medication to test for the elimination of the *B. pilosicoli* infection as it was a clinical problem in the field, rather than a clinical trial and costs to the farmer were to be kept to a minimum.

Results

The production data from an earlier flock, which had not been treated with tiamulin, was compared with the two younger treated flocks (see Table 1).

A similar number of birds were housed in each house but the untreated control flock (Flock A) was of a different breed, Lohmann Brown, rather than the two treated flocks, which were Bovans Goldline (Flocks B & C).

In egg production terms, the treated flocks showed a graded response to tiamulin treatment with the later treated flock (B – 45 & 60 weeks) showing a 5.2% and 5.6% improvement over the untreated flock (A) in egg numbers and total egg weight when adjusted to a 72 week age for comparison purposes and the flock treated at 25 & 45 weeks (C) giving a 9.8 and 9.7% improvement respectively. The average egg weights were almost identical and only minor differences in egg quality were recorded. The laying curve (see Figure 1) shows that flock C had a higher and longer peak laying percentage than the control A and B flocks but interestingly Flock B's performance tended to follow Flock C's performance following tiamulin treatment.

The average feed intake of Flock A (control) was lower, which affected egg production, however, somewhat surprisingly, the average hen bodyweight in Flock A (control) was higher than both the Goldline flocks, and Flock B would be considered very low for the breed at that age. Mortality in Flock A was substantially higher than Flock B and C by 6.73 and 8.59% respectively. This would have had a marked effect on the flock's overall performance when judged on a hen/housed basis.

To take into account the breed variation, the laying flocks were compared with their own breed standards. Flock A (untreated Lohmann flock) was compared to the Lohmann Brown standard performance data and laying curve for caged birds (see Table 2 and Figure 2). Flocks B and C, which received tiamulin treatment, were compared with the Bovans Goldline caged-bird standard production figures (see Table 3 and Figure 3).

Figure 2 demonstrated that Flock A started laying two weeks earlier than the standard and did not quite reach the normal peak of 94.5% but only 91.5% (-3%). However, the major difference was the subsequent performance upto 72 weeks where there was a major divergence in production and Flock A was down to 60% and the standard was 77.6% (-17.6%). Overall, Flock A produced 18.7 (-6%) fewer eggs than the standard and almost a kilo less in egg weight (-5.39%). Feed consumption was reduced by 2.03% in Flock A and the overall feed conversion ratio was worse by 3.56%. The most striking feature was the increased mortality rate of 13.84% in comparison with the flock standard of 5% (+8.84%).

Flock C (treated at 25 and 45 weeks of age) by contrast, mirrored fairly closely the Goldline standard laying curve. Peak production was 1% lower at 94%, but otherwise it followed the laying curve right upto 65 weeks when there was a small drop off in production by 72 weeks of 4%. Overall, Flock C laid 7.3 more eggs than the standard (+2.34%) and because the average egg weight was also higher, produced 0.8kg (+5.15%) higher egg weight. Feed consumption was noticeably higher by almost 3 kg and the FCR was therefore worse by 1.8%. The mortality rate was similar to the standard, only 0.25% higher.

Flock B, which was treated midway (week 45) and at week 60, showed a lower productivity than standard throughout the laying period. Peak laying percentage was down by 5% from standard and this continued for the rest of the laying period. Egg production was 1.94% lower than standard (-6.1 eggs) but average and total egg weight was higher by 3.19 and 1.19% respectively. Feed consumption was higher than standard and FCR was worse by 5.21%. Mortality was 2.22% higher than standard.

The comparative differences of the effects of the disease and treatments with the breed effect removed are summarised in Table 4. There was a graded response to treatment, with the untreated birds (Flock A) showing a 6% reduction in the number of eggs produced per hen housed to week 72, the late-treated birds (Flock B) showed a 1.94% drop and the early treated birds (Flock C) a 2.34% increase. The drop in egg production of 6% in the untreated group is closer to the 5% drop described by Swayne (2003). There was also a graded response to total egg weight. With regard to total feed intake there was less of a graded effect but more of a breed effect, with Goldline flocks consuming substantially more. There was no graded effect on FCR but both Flock A and B had a higher FCR variation than Flock C. Average bodyweight was very variable and breed related. Both the Goldline flocks were lower than the standard for the breed. There was a marked difference between the breeds at 72 weeks of lay with Lohmann meant to be 1.9kg (declining from a high of 1.95kg at 54 weeks) and Goldline 1.99kg bodyweight (increasing from 1.97kg at 54 weeks). There was a graded response to mortality with the untreated flock showing a substantially higher rate than the other two treated flocks. Peak laying percentage was lower in the untreated flock (A) as well as Flock B, which was not treated until much later at week 45, than the early treated Flock C (25 weeks of age), which suggests the infection could well be having an impact on this parameter.

Unfortunately, no statistical analysis of these data was considered possible due to the lack of replication of the flocks and treatments.

Discussion

Brachyspira pilosicoli is meant to be associated with a mild intestinal infection in laying birds with a drop of 5% in egg production and no impact on mortality (Swayne, 2003). However, if the condition is undiagnosed and untreated and lasts for the full length of the laying period, the effect seen was a 6% reduction in egg production and an increase of mortality by 8.84%. The mortality did not appear to be directly related to the *B. pilosicoli* infection, because it appeared to be associated with peritonitis caused by *E. coli* but might be indirectly, associated with the chronic debilitating stress of a chronic intestinal infection. It was not thought to be associated with the *M. synoviae* infection, although *E. coli* infections are commonly secondary to mycoplasmal infections, because this high level of mortality was not seen in other flocks, which also had *M. synoviae* infections and were controlled in the main by chlortetracycline medication. These factors demonstrate the chronic insidiousness of *B. pilosicoli* infections and highlight, that where there is a low grade level of diarrhoea, poor production and an increased mortality, that spirochaetosis should be considered as a differential diagnosis.

Because chlortetracycline at 600ppm in feed had no effect on the condition, in spite of repeated use, tiamulin was considered as the next suitable alternative in layers, as it also has a zero withdrawal period for eggs in the UK. Tetracycline appears to have a moderate activity in-vitro against *B. pilosicoli* with an MIC₉₀ below 5µg/ml (Hampson and Stephens, 2002), but as gut pharmacokinetic and pharmacodynamic data is not available for chickens a suitable microbiological breakpoint could not be determined and

it is suspected that the organism was resistant to chlortetracycline. The dose of tiamulin used (12.5mg/kg bodyweight) was lower than the one indicated for mycoplasma treatment in the UK (25mg/kg bodyweight) and by Stephens and Hampson (2002b) but higher than the level used in pigs for *B. hyodysenteriae* infections (8.8mg/kg bodyweight). There is also no data available for gut pharmacokinetics of tiamulin in chickens to establish a breakpoint, but a positive clinical response was achieved. Unfortunately, no faecal samples were taken directly after treatment to determine if the organism had been eliminated from the gut, as it was a field case. However, previous work by Stephens and Hampson (2002b) showed that indeed the organism was promptly removed from the birds following treatment and did not return within the four week observation period. At necropsy the organisms were also not recovered, unlike in the untreated controls. The birds were kept in cages in this field study, which tend to reduce faecal contamination and reduce the opportunities for reinfection, unlike free-range or barn-reared flocks, but a further application of tiamulin was given some months after the initial treatment as a precaution against re-infection.

The cause of the spread of the disease from flock to flock on this multi-age site was not established but thought to be most likely caused by mechanical transmission probably by flies but mice or other carriers could be involved. It was not affected by the season and continued all year round. Bano *et al* (2005) also reported that it was more common (86%) in older birds greater than 40 weeks of age than birds between 20-40 weeks of age (47%) and this was also found by Stephens and Hampson (1999). Birds kept in sheds with a deep pit system for droppings had a prevalence of 89% in comparison with a conveyor belt system and frequent removal with 58%.

It is interesting that two recent surveys in Australia and Italy (Stephens and Hampson, 1999; Bano *et al*, 2005) have shown a much higher prevalence of the potentially pathogenic spirochaetes, *B. pilosicoli* and *B. intermedia* than previously considered by the Dutch study (Dwars *et al*, 1989) and this could be explained by differences in the techniques used. Dwars *et al* (1989) used a direct fluorescent antibody test (FAT) on samples sent into the laboratory, whereas the others used a faecal sampling and culture technique followed by a polymerase chain reaction (PCR) test for isolate identification purposes. Both the Australian and Italian surveys showed that the prevalence of spirochaetes was about 70% (68-72%) and approximately half of those farms had pathogenic isolates of *B. pilosicoli* and *B. intermedia* (34-31% respectively). Swayne (2003) reported that the potential economic impact of the disease has not been estimated. However, in the UK, with a national flock of approximately 30 million hens and potentially 30% of flocks infected and they have a depression of egg production of 5% and an egg value of 3p/egg, then a figure of approximately £14 million can be estimated as the potential loss to the UK commercial laying industry or 1.5% of production.

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Table 1. Comparative production data for an infected untreated flock (A), an infected flock treated with tiamulin at 45 and 60 weeks of age (B) and a flock treated at 25 and 45 weeks of age (C)

Parameter	Flock A Infected Untreated	Flock B Treated Week 45 & 60	Flock C Treated Week 25 & 45	Comparison Flock C/A (%)
No of birds housed	12402	12032	12156	
Breed	Lohmann	Goldline	Goldline	
Eggs/hen housed	290.53	304.88	320.33	10.26
Eggs/HH at 72 weeks*	291.75	306.92	320.33	9.8
Egg weight (kg) at 72 weeks	18.59	19.63	20.40	9.7
Ave egg weight (g)	63.71	63.96	63.68	-0.05
Feed consumption/day (g)	111.285	119.954	120.432	8.2
Total feed 20-72 weeks (kg)	40.50	43.66	43.83	8.2
Feed/egg conversion ratio	2.179	2.220	2.148	-1.42
Ave bodyweight (kg) at sale	1.935	1.79	1.90	-1.3
Mortality (%)	13.84	7.11	5.25	-8.59
Peak laying (%)	91.5	90	94	2.5
Egg quality (%)				
V large	4.93	6.00	5.32	0.39
Large	40.27	40.36	42.05	1.78
Medium	38.08	34.12	34.33	-3.75
Small	4.94	3.56	4.35	-0.59
Seconds	11.78	15.96	13.94	2.16

* Adjusted to 72 weeks of production for flock comparison data

Table 2. Comparative production data for a *B. pilosicoli* infected untreated flock (A) and the Lohmann Brown caged hen standard to 72 weeks of age

Parameter	Flock A Infected Untreated	Lohmann Brown standard figures to 72 weeks	Comparison flock A / flock standard (%)
Breed	Lohmann	Lohmann	
Eggs/HH at 72 weeks*	291.75	310.4	-6.0
Egg weight (kg) at 72 weeks	18.59	19.65	-5.39
Ave egg weight (g)	63.71	63.30	0.65
Feed consumption/day (g)	111.285	113.600	-2.03
Total feed 20-72 weeks (kg)	40.50	41.35	-2.06
Feed/egg conversion ratio	2.179	2.104	3.56
Ave bodyweight (kg) at sale	1.935	1.900	1.84
Mortality (%)	13.84	5	8.84
Peak laying (%)	91.5	94.5	-3.0

* Adjusted to 72 weeks of production for flock comparison data

Table 3. Comparative production data for tiamulin-treated flocks (B & C) and the Bovans Goldline caged hen standard to 72 weeks of age

Parameter	Flock B Treated Week 45 & 60	Flock C Treated Week 25 & 45	Bovans Goldline standard figures to 72 weeks	Comparison flock B / Goldline standard (%)	Comparison flock C / Goldline standard (%)
Breed	Goldline	Goldline	Goldline		
Eggs/HH at 72 weeks*	306.92	320.33	313	-1.94	2.34
Egg weight (kg) at 72 weeks	19.63	20.40	19.40	1.19	5.15
Ave egg weight (g)	63.96	63.68	61.98	3.19	2.74
Feed consumption/day (g)	119.954	120.432	112.362	6.76	7.18
Total feed 20-72 weeks (kg)	43.66	43.83	40.90	6.75	7.16
Feed/egg conversion ratio	2.22	2.148	2.110	5.21	1.8
Ave bodyweight (kg) at sale	1.79	1.90	1.99	-10.05	-4.74
Mortality (%)	7.11	5.25	5.0	2.11	0.25
Peak laying (%)	90.0	94.0	95.0	-5.0	-1.0

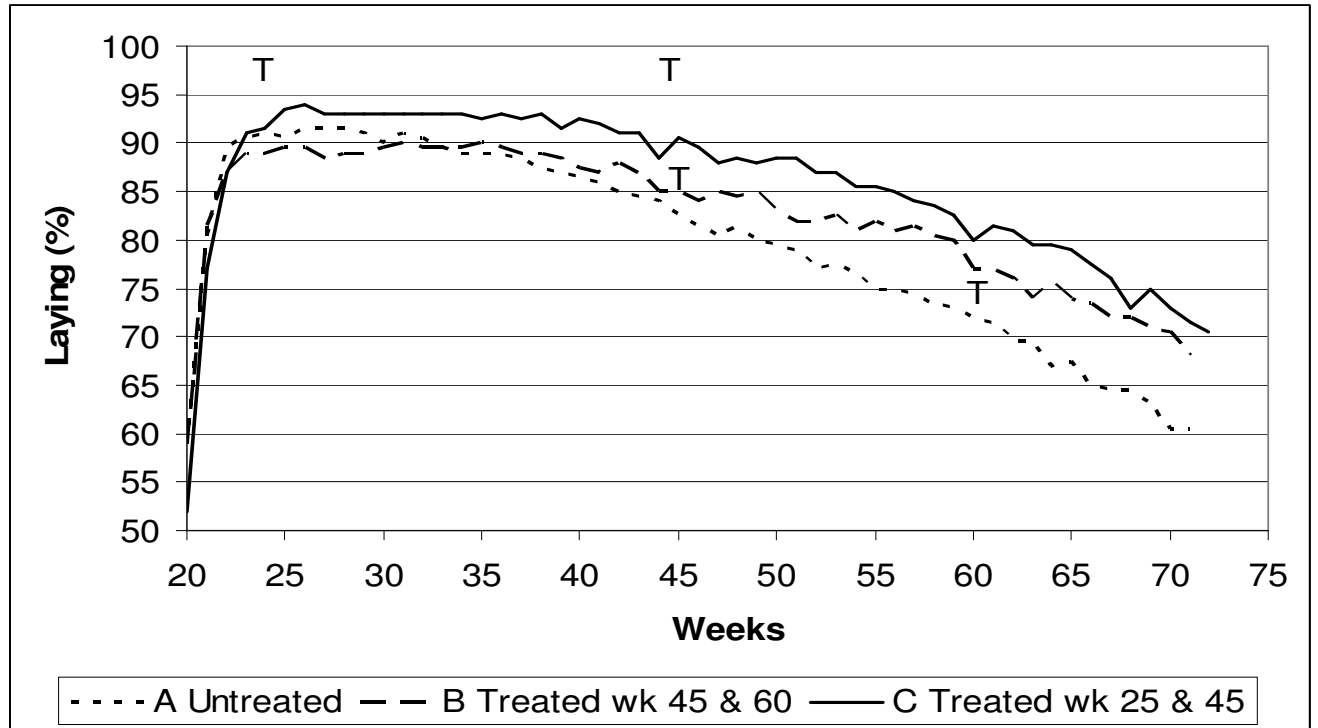
* Adjusted to 72 weeks of production for flock comparison data

Table 4. Comparison of individual flock's variations (%) from their breed standard

Parameter	Flock A Infected Untreated	Flock B Treated Week 45 & 60	Flock C Treated Week 25 & 45
Breed	Lohmann	Goldline	Goldline
Eggs/HH at 72 weeks*	-6.0	-1.94	2.34
Egg weight (kg) at 72 weeks	-5.39	1.19	5.15
Total feed 20-72 weeks (kg)	-2.06	6.75	7.16
Feed/egg conversion ratio	3.56	5.21	1.8
Ave bodyweight (kg) at sale	1.84	-10.05	-4.74
Mortality (%)	8.84	2.11	0.25
Peak laying (%)	-3.0	-5.0	-1.0

* Adjusted to 72 weeks of production for flock comparison data

Figure 1. Comparative laying curves of an infected untreated flock (A), an infected flock treated with tiamulin at 45 and 60 weeks of age (B) and a flock treated at 25 and 45 weeks of age (C)



Key: T – Tiamulin treatment flocks B and C

Figure 2. Comparative laying curves of the infected untreated flock (A) and the Lohmann Brown standard

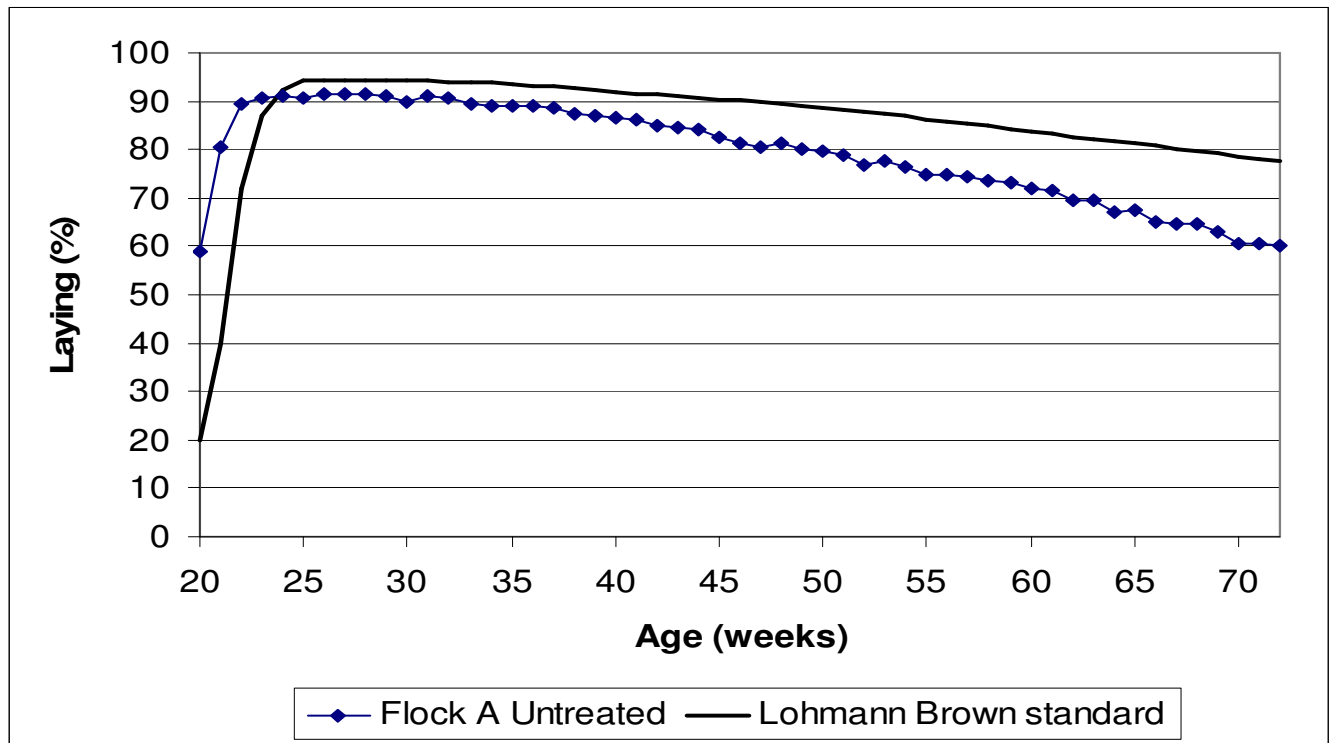
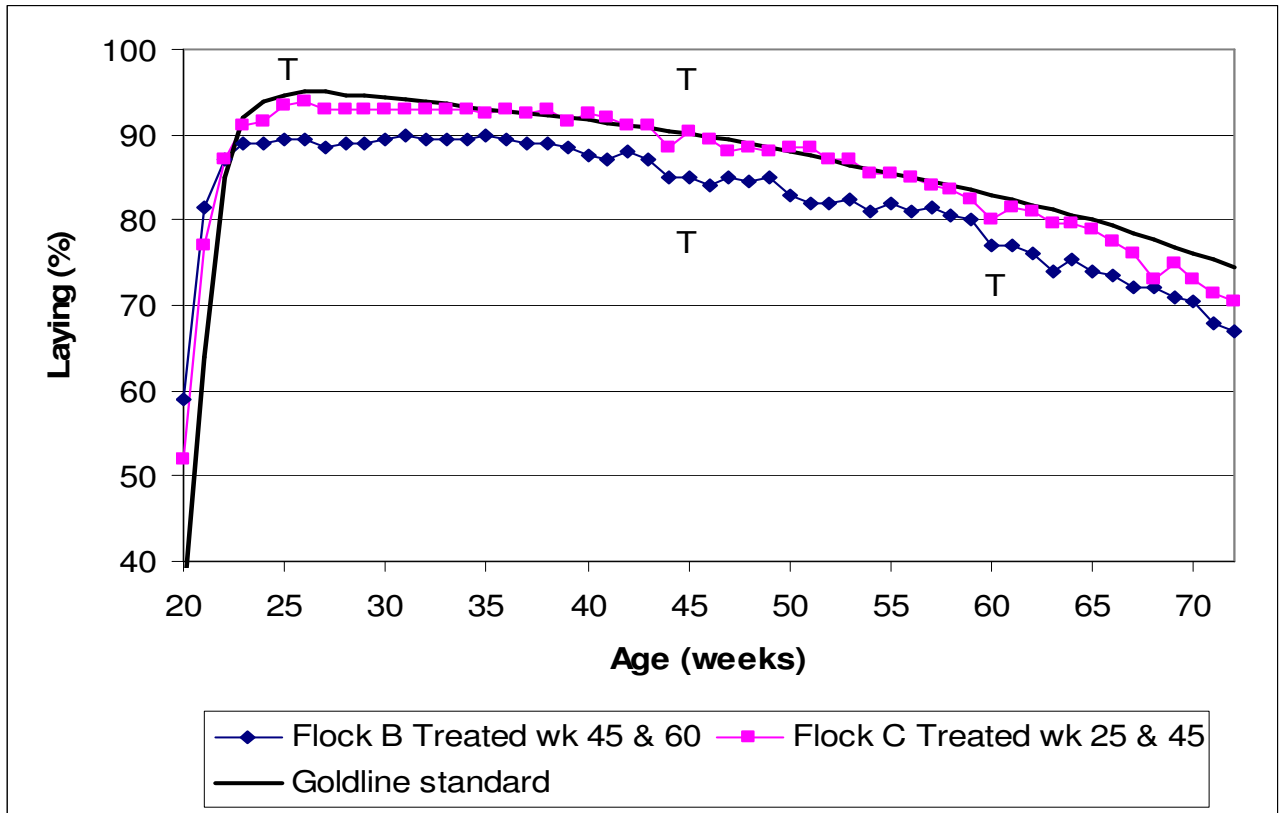


Figure 3. Comparative laying curves of the tiamulin-treated flocks (B & C) and the Bovans Goldline standard



Key: T – Tiamulin treatment flocks B and C

Pre-proof Only