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Comparison Between a Live, Attenuated Anticoccidial Vaccine and an Anticoccidial Ionophore, on Performance of Broilers Raised with or without a Growth Promoter, in an Initially *Eimeria*-Free Environment

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Waldenstedt L, Lundén A, Elwinger K, Thebo P, Uggla A: Comparison between a live, attenuated anticoccidial vaccine and an anticoccidial ionophore, on performance of broilers raised with or without a growth promoter, in an initially *Eimeria*-free environment. Acta vet. scand. 1999, 40, 11-21. – An experiment was carried out to study the effects of vaccination with Paracox®, a live, attenuated vaccine against avian coccidiosis, on broilers isolated from extraneous *Eimeria* parasites. The study involved 3200 broiler chickens raised in floor pens similar to commercial conditions, but in an initially *Eimeria*-free environment. Forty percent of the chickens were vaccinated at 3 days of age and given either a basal unmedicated feed or a feed supplemented with the feed antibiotic virginiamycin. Unvaccinated birds were given either the basal feed or feed supplemented either with virginiamycin or the anticoccidial ionophore narasin. At slaughter at 36 days of age vaccinated birds had a lower live weight than non-vaccinated birds. The difference was 4.6% in unmedicated, and 6.0% in virginiamycin medicated chickens. Feed conversion ratio at slaughter was 2.5% higher for unmedicated vaccinated birds, and 1.3% higher for virginiamycin medicated vaccinated birds, compared to respective non-vaccinated groups. There was no significant difference in overall performance of unvaccinated birds given narasin as compared to virginiamycin. At 10 days post vaccination vaccinated birds had a higher number of *Clostridium perfringens* in the caeca, but there was no difference thereafter. Throughout the experiment, caecal clostridial counts were considerably higher in vaccinated unmedicated birds than in unvaccinated birds given narasin. The number of oocysts shed in the vaccinated groups was very low, but during a subsequent challenge with *E. maxima* and *E. tenella* the birds' immunity was found to be satisfactory.

vaccination; coccidia; parasites; Clostridium perfringens; Paracox®

Introduction

Coccidiosis is a significant health and performance problem in modern broiler production. So far, control of the disease has mainly focused on the development and use of chemotherapeutic drugs, but the increasing appearance of drug resistant parasite strains and growing costs associated with development of new anticoccidial drugs has stimulated a search for alternatives to chemotherapy. Immunological control seems to be the major practical al-

ternative to chemotherapy for controlling coccidiosis. Live, attenuated vaccines have been developed, and proven to be both efficacious and commercially feasible (*Shirley & Long* 1990, *Williams* 1992). These vaccines stimulate natural immunity without risk of outbreaks of clinical coccidiosis. Due to costs of the vaccines interest has so far mainly been focused on vaccination of layers and heavy breeders, and not so much on broilers. Therefore, relatively little is known about the effect of vaccination on broiler performance, especially when the chickens are raised without feed antibiotics.

In Sweden, routine use of feed antibiotics for domestic animals has been prohibited by law since 1986. However, in commercial broiler production coccidiostats are still routinely used on veterinary prescription to prevent coccidiosis. By agreement between authorities regulating Swedish broiler production, mainly one ionophore coccidiostat (narasin) has been used for the last 10 years. Besides protecting against coccidiosis, narasin has been shown to have general growth promoting as well as antibacterial effects, especially against *Clostridium perfringens* (*Kondo* 1988, *Elwinger et al.* 1992; 1996).

C. perfringens is part of the normal bacterial flora in the gastrointestinal tract of poultry. Normally the number of these bacteria in the intestine is low, but under certain conditions they may multiply and cause enteric disease. *C. perfringens* type A and type C has been shown to be causal agents of necrotic enteritis (NE), a serious chicken disease throughout the world (*Ficken* 1991). Many antibiotic substances used as growth promoters inhibit the growth of *C. perfringens,* which can prevent NE (*Dutta & Devriese* 1980, *Kondo* 1988). Since some ionophore coccidiostats also have Gram-positive antibacterial activity they are likely to play a role in the control of *C. perfringens.* Therefore, the replacement of ionophore coccidiostats by vaccination may increase the risk of NE when no feed antibiotic is used.

Consequently, comprehension of the chickens' response to anticoccidial vaccines is an important field of research both from a scientific and a commercial point of view, if vaccination is to become an alternative to anticoccidials in future broiler production. So far, Paracox® (Shering-Plough, Middlesex, UK) is the only licensed vaccine against avian coccidiosis in Sweden. It is a live, attenuated vaccine comprising a stabilised suspension of sporulated oocysts of the 7 species of *Eimeria* (*E. acervulina, E. brunetti, E. maxima* (2 strains), *E. mitis, E. necatrix, E. praecox* and *E. tenella*) that parasitize the domestic fowl (*Williams* 1992).

The aim of the experiment reported here was to study the effects of Paracox® on performance of broilers given a diet with or without feed antibiotics in comparison with chickens fed an anticoccidial ionophore supplemented diet. To investigate the influence of the vaccine itself the study was carried out in an environment free from extraneous *Eimeria* parasites.

Materials and methods

The experiment included 3200 unsexed Ross broiler chickens delivered on day of hatching from a commercial hatchery (Kronfågel, Väderstad, Sweden). The experiment was carried out during October to December 1995. The chickens were reared in a building with 24 floor pens (each 11m^2) with wood shavings used as litter. Half the number of pens were supplied with nipple drinkers, the rest with bell drinkers. The experiment consisted of 5 experimental treatments according to Table 1. Each treatment consisted of 4 replicates (pens) each with 160 chickens at the beginning of the experiment.

The composition of the basal diet was similar to a commercial Swedish broiler diet (Table 2).

Table 1. Experimental design.

Treatment	Vaccination	Feed additives	mg/kg
Control	no	none	–
Control	yes	none	–
Antibiotic	no	virginiamycin	20
Antibiotic	yes	virginiamycin	20
Coccidiostat	no	narasin	70

The diets were manufactured at the Agricultural University feed mill. The cereals were ground using a hammer mill with a 5 mm screen, and the diets were steam-pelleted at 75 °C using a 3 mm die. During the first week the pellets were crushed to smaller particles. Virginiamycin (Stafac®, Pfizer, New York, USA) and narasin (Monteban®, Elanco, Indianapolis, USA) were incorporated at 20 and 70 mg/kg of active substance, respectively, and given from day one. Virginiamycin was given until slaughter, but narasin was withdrawn 5 days before slaughter. Analysis of the pelleted experimental diets confirmed the concentration of the test substances (69.2 mg narasin and 22.7 mg virginiamycin per kg feed).

Vaccination with Paracox® was performed on one occasion at 3 days of age according to the manufacturer's recommendation. Chickens were deprived of water for 2 h before the vaccine was administered via the water in bell type drinkers. In pens with nipple drinkers, bell type drinkers were supplied during vaccination. In order to avoid spread of the vaccine strains to non-vaccinated birds, vaccinated groups were isolated from the other groups by empty pens and walls of plastic sheet. Separate working clothes and tools were used for each area.

The chickens were weighed on a pen basis on days 1, 13, 28 and at slaughter at 36 days of age. Accumulated feed intakes (FI) and feed conversion ratios (FCR) were calculated at these ages.

Table 2. The basal diet.

Ingredients	%	Calculated nutrient content	%
Wheat	51.00	ME, MJ/kg	12.0
Barley	13.00	Crude protein (analysed)	19.7
Oats	10.00	Lysine	1.15
Soybean meal	14.00	Methionine	0.51
Rapeseed meal 200	1.00	Meth + Cys	0.85
Fish meal	3.65	Threonine	0.66
Meat meal	2.00	Crude fat	4.4
Vegetable fat (Akofeed, standard[1])	2.10	Linolic acid	1.17
Vitamin- and mineral premix	1.00	Ca	0.98
Calcium carbonate	0.50	P, total	0.70
Sodium chloride	0.20	P, available	0.45
Dicalciumphosphate	1.00	K	0.66
Methionine	0.20	Na	0.15
Lysine-HCl	0.32	Cl	0.24
Enzymes (Biofeed plus)	0.03		

[1] Akofeed standard (Karlshams Crushing & Feed AB, Karlshamn, Sweden) is obtained during vegetable oil processing, and contains vegetable fatty acids (17% linoleic acid).

FCR included the weights of chickens removed from the experiment due to mortality, or for intestinal lesion scoring or bacterial counts. Water intake was recorded daily per pen. Numbers of chickens with droppings attached to the cloaca down (sticky droppings) were recorded at 7 days of age. Dry matter content of the litter was determined on a sample (of approximately 500 g) taken in the middle of each pen when the birds were 3 and 5 weeks old.

Twice weekly, litter samples were collected from the same 5 places in every pen and then pooled to give one sample (of approximately 200 g) per pen. Out of these, double samples were analyzed for number of oocysts per gram of faeces (OPG) using a modified McMaster technique. A 20-30 g portion of the sample was mixed with tap water at a 1:15 ratio. After homogenization the suspension was filtered through gauze and five 2 ml aliquots were withdrawn and pooled in a tube. After centrifugation the pellet was resuspended in 10 ml saturated sodium chloride solution and the OPG determined as described by *Taylor et al.* (1995). On days 13, 21, 27 and 34, two chickens per pen were killed for examination of intestinal lesions and scored according to the method of Johnson & Reid (1970). Analysis of *C. perfringens* in the caeca was also performed on these chickens. Thus, a total of 160 chickens (4 time points×2 chickens×20 pens) were killed and examined. For analysis of *C. perfringens* one caecum per chicken was removed immediately after opening the abdomen using aseptic techniques, transferred to a sterile Petri dish and stored for a maximum of 2 h at 4 °C before microbiological examination. Determination of *C. perfringens* was carried out according to the Nordic Committee on Food Analyses (1985), but excluding the use of sporulation medium for identification.

In order to evaluate the immunity induced by the vaccination, 6 chickens per treatment, excluding groups given coccidiostats (in total 24 birds), were randomly selected and removed from the experiment on day 36. The chickens from each treatment were placed on clean wood shavings in separate boxes (0.75×1.2 m). Chickens were fed the same diet as prior to the challenge inoculation. On day 37, the birds were individually weighed and inoculated via the crop with a suspension of 10 000 plus 2 000 sporulated oocysts of *E. maxima* and *E. tenella,* respectively. The oocysts, originally isolated from Swedish broiler and layer flocks (*Thebo et al.* 1998), were recovered after propagation in 3-week-old *Eimeria*-free chickens (*Shirley* 1995) and sporulated in 2% potassium dichromate at 4 °C before inoculation. Oocysts per gram of faeces (OPG) on days 6, 7 and 8 post inoculation (dpi) were determined. Weight gain was measured on 8 dpi, prior to the chickens were being killed.

The remaining chickens were slaughtered at 36 days of age. Autopsies were performed on all chickens that died during the experiment. No detectible antibodies against infectious bursal disease virus (IBDV) were detected in blood samples collected from chickens at slaughter.

Statistical analyses

Statistical analyses were based on analysis of variance, using the General Linear Model procedure of SAS® (*SAS Institute* 1994). All main effects were considered fixed. Relative frequencies for mortality and sticky droppings were angularly transformed before statistical analyses according to *Snedecor & Cochran* (1968). Performance data were corrected for differences between water systems. The pen of chicks served as the experimental unit for all data except intestinal lesion scores.

Results

Chicken performance is shown in Table 3. Vaccinated birds had a lower live weight than the

Table 3. Effect of vaccination and feed additives on production performance of broiler chickens[1].

		Vaccinated		Non-vaccinated			Statistical p-values				
	Age, days	Control	Virginiamycin	Control	Virginiamycin	Narasin	Vacc.[2]	Antib.[3]	Treatm.[4]	(LSD)	CV,%
Live weight, g	13	302	398	309	413	392	0.01	0.001	0.001	(11.5)	2.1
	28	1140	1353	1163	1400	1366	0.02	0.001	0.001	(42.1)	2.2
	36	1711	1919	1794	2041	2007	0.001	0.001	0.001	(54.9)	1.9
Acc. feed intake, g	13	356	425	356	439	422	0.14	0.001	0.001	(13.5)	2.2
	28	1665	1958	1671	1954	1928	0.96	0.001	0.001	(78.3)	2.8
	36	2759	3104	2808	3208	3147	0.04	0.001	0.001	(95.4)	2.1
Feed conversion	13	1.18	1.07	1.15	1.06	1.08	0.07	0.001	0.001	(0.03)	1.6
ratio	28	1.46	1.44	1.44	1.39	1.41	0.003	0.02	0.004	(0.03)	1.4
	36	1.61	1.59	1.57	1.57	1.57	0.01	0.49	0.08		1.5
Water intake, ml	13	689	782	671	763	695	0.61	0.03	0.21		9.9
	28	2835	3132	2777	3103	2982	0.43	0.001	0.002	(159)	3.5
	36	4582	4948	4507	5017	4804	0.96	0.001	0.001	(203)	2.8
Water/feed ratio	13	1.93	1.89	1.88	1.76	1.64	0.20	0.45	0.21		10.0
	28	1.70	1.60	1.70	1.60	1.54	0.07	0.07	0.07		4.5
	36	1.66	1.59	1.61	1.57	1.52	0.02	0.11	0.03	(0.08)	3.2
Acc. mortality,%	13	1.8	2.4	2.4	1.9	2.0	0.67	0.85	0.94		52.5
	28	3.2	3.4	3.0	2.3	3.4	0.36	0.89	0.90		30.8
	36	3.5	3.8	3.2	2.8	3.6	0.36	0.98	0.91		26.7
C. perfringens, $\log_{10}$	13	6.90	1.65	6.72	0.59	1.43	0.05	0.001	0.001	(0.88)	16.8
	21	7.14	1.47	6.97	2.06	1.92	0.68	0.001	0.001	(1.51)	25.3
	27	6.67	1.87	6.88	1.98	1.75	0.87	0.001	0.002	(2.95)	50.8
	34	6.37	1.77	5.49	1.64	3.54	0.54	0.001	0.003	(2.40)	42.1

[1] Vaccination at 3 days and slaughter at 36 days of age.
[2] Comparison between vaccinated and unvaccinated groups, excluding groups given coccidiostat.
[3] Comparison between control groups and groups given feed antibiotics.
[4] Differences between treatments, and Least Significant Difference for $p<0.05$.

corresponding non-vaccinated birds. The difference was 4.6% in unmedicated birds, and 6.0% in virginiamycin treated chickens. There was no difference in FI due to vaccination until 36 days of age, when the FI of unmedicated and virginiamycin treated birds were 1.7% and 3.2% lower than those of the corresponding non-vaccinated controls ($p<0.04$). FCR were significantly higher for vaccinated birds, 2.5% for unmedicated and 1.3% for virginiamycin treated birds, at 36 days ($p<0.01$). Vaccination had no effect on accumulated water intake (WI), but the water:feed ratio (WFR) was 3.1% and 1.0% higher for unmedicated and virginiamycin treated vaccinated chickens, respectively, at 36 days ($p<0.02$).

Addition of virginiamycin increased LW by 12.2% in vaccinated birds, and by 13.8% in unvaccinated birds. Virginiamycin also increased FI by 12.5% in vaccinated and 14.2% in unvaccinated birds. At 13 days, the FCR of vaccinated birds fed virginiamycin was 9.3% lower than that of vaccinated unmedicated birds, but there was no significant difference at 28 or 36 days. In non-vaccinated birds, virginiamycin addition improved FCR by 7.8% and 3.5% at 13 and 28 days, respectively, but had no effect at 36 days. Virginiamycin also enhanced WI by 10.8% in vaccinated, and by 11.1% in unvaccinated chickens, but had no significant influence on WFR. There was no difference in performance of non-vaccinated chickens given coccidiostat compared with those given virginiamycin, except at 13 days when chickens given coccidiostat had a lower LW and FI than chickens given virginiamycin. No interaction between virginiamycin and vaccination was found.

At 13 days vaccinated chickens had a higher number of *C. perfringens* ($p<0.05$) in the caeca, but thereafter there was no difference (Table 3). Both vaccinated and non-vaccinated chickens given virginiamycin had significantly lower numbers ($p<0.001$) of *C. perfringens* than the control groups. Neither was there any significant difference in this aspect between the unvaccinated groups given coccidiostat and those given feed antibiotic. Overall, groups given either of the additives had lower numbers of *C. perfringens* than control groups (Table 3).

Intestinal examinations revealed a slight hyperaemia mainly in the small intestine of vaccinated chickens and occasional minor lesions, which were below the lowest possible score (1) on the scale set by *Johnson & Reid* (1970). Vaccinated chickens shed low numbers of oocysts, Fig. 1. During the last week (from 31 days of age), low numbers of oocysts were detected in samples from 5 of the non-vaccinated groups.

Average dry matter content in the litter bed was 81% ± 2% (SD) at 3 weeks, and 66% ± 8% at 5 weeks. There was no significant difference between treatments, either at 3 or 5 weeks. At 7 days, 0.9% (30 chickens) of all birds, had sticky droppings without any significant differences between treatments. Mortality was on average

Table 4. Weight gain after challenge infection at 37 days of age (n=6).

Additives	Vaccination	Average weight at challenge (g) ± SD	Weight gain 8 d.p.i	
			g	%
None	Yes	2114 ± 85	481	28.4
None	No	2105 ± 165	326	19.1
Virginiamycin	Yes	2242 ± 273	537	30.9
Virginiamycin	No	2085 ± 211	226	14.3

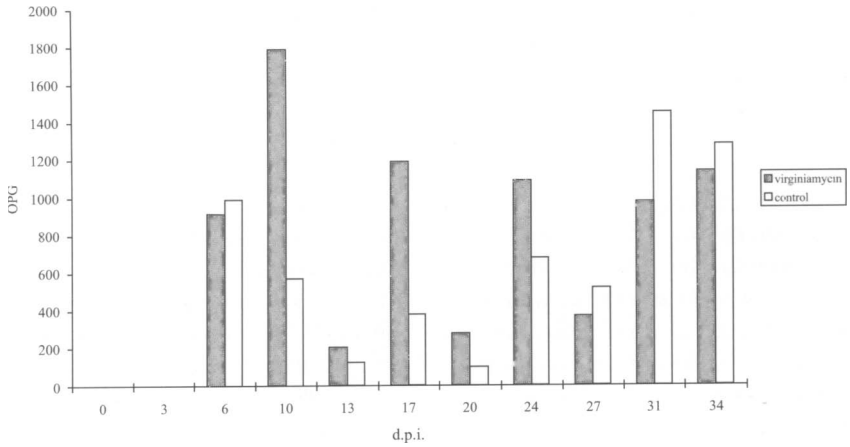

Figure 1. Oocysts per gram faeces (OPG) in vaccinated birds.

3.4%, and not affected by either vaccination or feed additives (Table 3). The most frequent cause of death was acute death syndrome, and the spectrum of post mortem diagnoses was similar for the different treatments.

After the challenge infection, weight gain for vaccinated chickens was 29.7%, compared with 16.7% for non-vaccinated chickens ($p<0.002$) (Table 4). Oocyst output after challenge is shown in Fig. 2. After challenge, OPG in vaccinated birds were at most 8% of that of unvaccinated, and oocysts were found 1 day later in vaccinated birds than in unvaccinated birds.

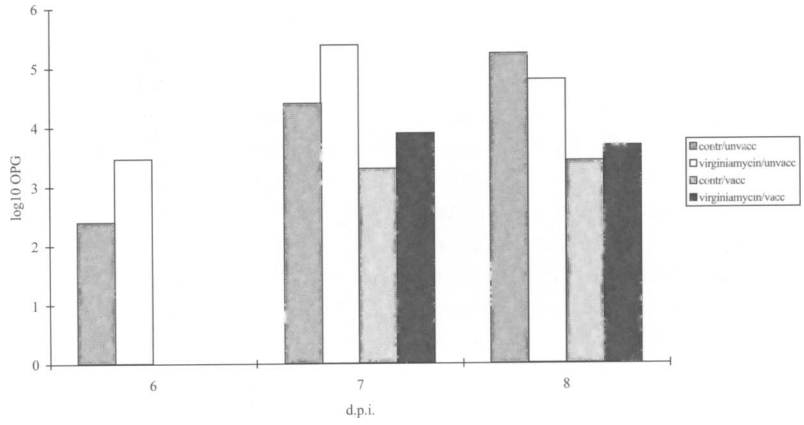

Figure 2. Oocysts per gram faeces (OPG) after challenge inoculation.

Discussion

On the whole, chicken performance was good in all groups. However, vaccinated chickens had lower live weights and higher feed conversion ratios than non-vaccinated birds on all recording occasions, and accumulated feed intake of vaccinated chickens was moderately depressed between 28 and 36 days, independently of virginiamycin treatment. This contrasts results reported by *Williams* (1994), who found no negative effects of vaccination on live weight gain, feed intake or feed conversion efficiency in avoparcin treated chickens isolated from extraneous coccidial infection. The negative effect of the vaccination recorded in this study is difficult to explain. Intestinal hyperaemia and minor lesions were observed in vaccinated birds, and although these reactions appeared insignificant they may have had some impact on chicken growth. However, *Williams* (1994) found occasional lesions in vaccinated chickens, but no adverse effects on performance, supporting other observations (*Conway et al.* 1990, *McKenzie et al.* 1989) that correlation between intestinal lesions and growth is not consistent in immune birds.

It has been shown that activation of the immune system can result in reduced growth rates in chickens. *Klasing et al.* (1987) reported that a growth-depressing effect could be induced by administration of a number of non-infectious inflammatory agents. They concluded that this effect was correlated with the immunogenic strength of the agents and the duration and vigour of the immune responses. Decreased feed intake was identified as the main factor, but also less effective intermediary metabolisms contributed to the reduction in performance. However, a comparison between such experimentally induced immunological stress and the presumably mild immune reactions triggered by vaccination with avirulent vaccinal *Eimeria* strains is difficult, and it is not impossible that other unknown factors contributed to the difference between the performances of vaccinated and non-vaccinated animals observed in the present study.

Eimeria infections have been shown to predispose chicks to clostridial growth (*Baba et al.* 1988). Since feed antibiotics, and to a certain extent, some ionophore coccidiostats, depress the growth of these bacteria, it was suspected that, in a situation where feed antibiotics were not used, the substitution of an ionophore coccidiostat with a vaccine might increase the occurrence of *C perfringens.* This was also the case in the present experiment where vaccinated birds not given any supplement had a markedly higher number of *C. perfringens* in their caeca as compared to birds given coccidiostat, which thus would increase the risk of necrotic enteritis. The considerable difference in growth rate between birds given the basal and supplemented diets may also be related to the occurrence of *C. perfringens* (*Stutz & Lawton* 1984). This shows that if the use of coccidiostats is to be replaced by vaccination, clostridial numbers in the chicken intestine have to be controlled by other means. After vaccination with Paracox® a slight increase in numbers of *C. perfringens* in the caeca was noted during the first part of the rearing period. However, this relatively small increase is probably without any practical importance, neither with regard to differences in growth between vaccinated and unvaccinated birds, nor the risk of necrotic enteritis.

Litter bed quality is an important factor involved in broiler performance. Coccidiostats may, in various ways, influence water consumption and water:feed ratio which affects litter bed quality. Narasin usually has a favourable effect on litter quality by decreasing water:feed ratio (*Elwinger et al.* 1994). It was therefore also of interest to study the effect of vaccination on litter condition. In the present experiment vacci-

nation did not affect water intake, but as a consequence of the lower feed intake the water:feed ratio increased. Yet, litter quality in pens with vaccinated birds and non-vaccinated narasin treated birds was similar. In the study by *Williams* (1994) the water consumption (and the water:feed ratio) was lower for the vaccinated birds in comparison to nicarbazin-treated birds, which was reflected in an improved litter quality for vaccinated birds. However, differences between different feed antibiotics and coccidiostats could be the reason for the difference between these results.

Despite the precautions taken, 5 of the unvaccinated groups became infected with coccidia and shed low numbers of oocysts. The affected animals did not show any clinical symptoms. Spread of vaccine strains seems to be the most likely reason for this, since no *Eimeria* parasites had previously been detected in the experimental area. Contamination occurred late in the experiment and did not affect the live weights of contaminated groups compared with the corresponding uncontaminated groups.

When *Williams* (1994) studied the effects of Paracox® in broilers isolated from extraneous coccidial infection, the numbers of oocysts in the litter peaked at 5 or 12 days post vaccination. Five days after vaccination oocyst counts were on average 12 000, and thereafter numbers declined until they became below the detection limit at some time between 26 and 40 days after vaccination. In our experiment, oocyst numbers were markedly lower. Nevertheless, during the subsequent challenge with *E. maxima* and *E. tenella* the chickens' immunity was found to be satisfactory as judged by weight gain after the challenge infection. Oocyst shedding after challenge infection was higher in non-vaccinated birds, also verifying the efficacy of the vaccination.

In practical conditions, if coccidiostats were to be replaced by vaccination with Paracox® in Swedish broiler flocks, a comparison between vaccinated chickens raised without feed-additives and non-vaccinated chickens given coccidiostats is valid. When comparing live weights of unvaccinated chickens fed unmedicated or narasin medicated diets in an *Eimeria* free environment, *Elwinger et al.* (1992; 1994; 1996) and *Waldenstedt & Elwinger* (1995) observed increases in live weight in narasin-treated birds ranging from 5% to 11% at 6 weeks of age. In the present experiment unvaccinated chickens given coccidiostat had a higher (17%) live weight at 36 days than vaccinated chickens. Accumulated feed intake at 36 days were 14% higher for chickens given coccidiostat, but there were no differences in feed conversion ratio at that age. However, the present experiment was carried out in an environment free from extraneous virulent coccidia and other major pathogens, and depending on surrounding conditions the effects of substituting coccidiostats with a vaccine might differ from the present study. Factors that are likely to affect the results are general hygiene, coccidial infection pressure, occurrence of coccidiostat-resistant coccidia, efficacy of the vaccine, stress and general health.

To conclude, the results of the present study showed that the performance of vaccinated broilers was slightly impaired, irrespective of virginiamycin treatment, and that anticoccidial vaccination did not have any major effect on the number of *C. perfringens* in the caeca. Further studies are required to obtain a deeper understanding of the chickens' basal response to live coccidial vaccines.

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Sammanfattning

Effekten av ett levande koccidievaccin på produktionsresultaten hos slaktkycklingar, uppfödda med eller utan foderantibiotika, i en miljö fri från naturliga koccidier.

Effekterna av vaccinering med Paracox®, ett levande, attenuerat vaccin mot koccidios hos fjäderfä, studerades hos 3200 slaktkycklingar. Kycklingarna föddes upp på golvströbädd i en miljö fri från naturliga koccidier. Fyrtio procent av kycklingarna vaccinerades oralt via dricksvattnet vid 3 dagars ålder. Vaccinerade kycklingar fick antingen ett basfoder (12,0 MJ/kg, 19,7% råprotein) utan tillsatser (B), eller med tillsats av 20 mg virginiamycin/kg (V). Ovaccinerade kycklingar fick antingen foder B, foder V eller basfodret med tillsats av koccidiostatika (narasin 70 mg/kg) (N). Vid slakt vid 36 dagars ålder var de vaccinerade kycklingarnas levande vikt lägre än de ovaccinerades (4,6% för foder B och 6,0% för foder V). Även foderomvandlingsförmågan (kg foder/kg levande vikt) var sämre hos de vaccinerade kycklingarna (2,5% för foder B och 1,3% för foder V). Det var ingen skillnad i produktions- och hälsohänseende mellan kycklingar som fått foder V eller N. Antalet *Clostridium perfringens* i blindtarmarna var högre hos vaccinerade djur 10 dagar efter vaccinationen, men därefter påverkade vaccineringen inte förekomsten av dessa bakterier. De vaccinerade kycklingarna utsöndrade endast små mängder oocystor. Experimentell inokulering vid 37 dagars ålder av en mindre grupp kycklingar visade att en tillfredsställande immunitet hade utvecklats.

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