Impact of fenbendazole resistance in *Ascaridia dissimilis* on the economics of production in

turkeys

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ABSTRACT

Feed conversion efficiency is among the most important factors affecting profitable production of poultry. Infections with parasitic nematodes can decrease efficiency of production, making parasite control through the use of anthelmintics an important component of health management. In ruminants and horses, anthelmintic resistance is highly prevalent in many of the most important nematode species, which greatly impacts their control. Recently, we identified resistance to fenbendazole in an isolate of *Ascaridia dissimilis*, the most common intestinal helminth of turkeys. Using this drug-resistant isolate, we investigated the impact that failure to control infections has on weight gain and feed conversion in growing turkeys. Birds were infected on Day 0 with either a fenbendazole-susceptible or -resistant isolate, and then half were treated with fenbendazole (SafeGuard® Aquasol) at 4- and 8-weeks post infection. Feed intake and bird weight were measured for each pen weekly throughout the study, and feed conversion rate was calculated. Necropsy was performed on birds from each treatment group to assess worm burdens at weeks 7 and 9 post infection. In the birds infected with the susceptible isolate, fenbendazole-treated groups had significantly better feed conversion as compared to untreated groups. In contrast, there were no significant differences in feed conversion between the fenbendazole-treated and untreated groups in the birds infected with the resistant isolate. At both weeks 7 and 9, worm burdens were significantly different between the treated and untreated birds infected with the drug-susceptible isolate, but not in the birds infected with the drug-resistant isolate. These significant effects on feed conversion were seen despite having a rather low worm establishment in the birds. Overall, these data indicate that *A. dissimilis* can produce significant reductions in feed conversion, and that failure of treatment due to the presence of fenbendazole-resistant worms can have a significant economic impact on turkey production.

46 Furthermore, given the low worm burdens and an abbreviated grow out period of this study, the

47 levels of production loss we measured may be an underestimate of the true impact that

48 fenbendazole-resistant worms may have on a commercial operation.

49   **KEYWORDS**

50 *Ascaridia*, benzimidazoles, anthelmintic resistance, feed conversion, turkey

51   **1.INTRODUCTION**

Both helminth and protozoan parasites can impact poultry performance parameters such

as weight gain and/or feed conversion ratio (FCR) (Voeten, Braunius et al. 1988, Daş, Kaufmann

et al. 2010, Sharma, Hunt et al. 2019). Feed conversion, a measure of feed consumption per unit

of production accounts for approximately 70% of production costs, making it among the most

important factors affecting profitable production (Willems, Miller et al. 2013). A lower feed

conversion ratio (FCR) indicates that feed is being more efficiently utilized for growth. While

coccidia (*Eimeria* spp.) are well documented as important parasitic pathogens of poultry,

helminths generally receive much less attention. Several studies in chickens have shown that

infections with *Ascaridia galli* have a negative impact on both feed efficiency and egg quality

(Daş, Kaufmann et al. 2010, Stehr, Grashorn et al. 2019). However, less work has been done

investigating this issue in turkeys infected with *Ascaridia dissimilis.*

Ascaridia dissimilis is the most prevalent and one of the most important parasites of

turkeys, with up to 100% of a flock being infected (Yazwinski, Tucker et al. 2009). *Ascaridia*

eggs are capable of surviving the environmental extremes that are present in poultry houses and

may remain infective for periods exceeding six months, leading to a cycle of continuous

reinfection and environmental contamination with new eggs (Cauthen 1931, Tarbiat, Jansson et

al. 2015). Heavy infections may cause clinical disease such as diarrhea, intestinal blockage, and

enteritis, but most often infections are subclinical, only causing reduced feed efficiency (Ikeme

1971, Norton, Hopkins et al. 1992, Yazwinski, Tucker et al. 2002). Given the potential health

and production impacts of *Ascaridia*, as well as its near ubiquity, successful control will often be

important for profitable production.

Currently, in the United States, fenbendazole is the only available treatment approved by

the Food and Drug Administration for treatment of *Ascaridia* infections in poultry. Registration

studies of fenbendazole (SafeGuard®) in feed, at 1mg/kg body weight for 6 days, demonstrated

greater than 99% efficacy against *Ascaridia dissimilis* (United States Food and Drug

Administration 2000). In addition, a formulation of fenbendazole that is administered in water,

(SafeGuard® Aquasol), demonstrated a mean efficacy of 97.7% against *Ascaridia galli*, a closely

related parasite of chickens, that may also infect turkeys (United States Food and Drug

Administration 2018). On commercial turkey farms, treatments with fenbendazole are often

administered frequently, around every 4 weeks, which is an interval less that the prepatent period

A. dissimilis. These treatments are typically administered in either feed or water to the entirety of

the house. These means of drug delivery make accurate dosing challenging due to difficulty in

optimal delivery of the drug and variability in consumption. Both issues may lead to sub-

therapeutic levels of ingestion in some birds. In other livestock species, under-dosing is thought

to be an important factor influencing the development of drug resistance in nematode parasites

(Smith, Grenfell et al. 1999, Jackson and Coop 2000). A model investigating factors promoting

the development of anthelmintic resistance showed that repeated under-dosing acted as a strong

selector for resistance, since partially resistant heterozygotes were able to survive and reproduce

(Smith, Grenfell et al. 1999). The survival of heterozygotes led to a much more rapid increase in

the frequency of resistant homozygotes in the population as compared to full-dose treatments

that killed the heterozygotes with high efficacy. Under-dosing, combined with often intensive

use in production animals, may act as strong selectors for the development of anthelminthic

resistance in nematode parasites.

In many species of important livestock parasites, resistance to benzimidazoles is highly

prevalent (Kaplan 2004, Howell, Burke et al. 2008, Kaplan and Vidyashankar 2012). Though

reduced efficacy of fenbendazole was reported previously in *Ascaridia dissimilis*, (Yazwinski,

Tucker et al. 2013) resistance to fenbendazole in *A. dissimilis* was only recently confirmed for

the first time in a controlled efficacy study (Collins, Jordan et al. 2019). Following treatment

with fenbendazole, a field isolate of *A. dissimilis* (Sn) yielded an efficacy of 63.9%, whereas in

three other field isolates fenbendazole treatment yielded an efficacy of greater than 99%. Having

demonstrated fenbendazole resistance in a naturally occurring field isolate of *Ascaridia*

dissimilis, we wanted to measure the effects that resistant parasites may be having on production

parameters as a consequence of failed treatments.

106   **2.MATERIALS AND METHODS**

2.1 Turkeys and feeding

Four hundred and thirty-two, day old, Hybrid turkey poults were received from Prestage

Farms and housed at the Poultry Science farm at the University of Georgia. Birds were allowed

one week of acclimation before the study began. Water and feed were provided *ad libitum*. For

the first 6 weeks, birds were fed a starter ration with 26% protein, then a grower ration with 23%

protein was offered from weeks 6 to 9 (see Supplemental files 1 & 2 for the diet formulations).

2.2 Study Design

Birds were received on Day -7 and were assigned to 36 pens of 12 birds each based on

weight, minimizing differences in total weight between pens. 16 pens were infected with the

resistant isolate, 16 pens were infected with the susceptible isolate, and 4 pens served as

environmental controls. Groups were separated by floor to ceiling mesh curtains to prevent

movement of birds between pens. Feed was added into hanging feeders and the initial weight of

feeders for each pen was recorded. Each subsequent week, total bird weight for each pen and the

weights of feeders were recorded to determine the weight gain and feed consumed. The hanging

feeders were then refilled and an initial feeder weight for the next week was recorded. At weeks

7 and 8 post infection (p.i.), groups were culled to 10 and 9 birds respectively, to maintain

recommended stocking densities. The study was originally planned to continue for 16 weeks but

was terminated at week 9 due to inability of the facilities to properly contain turkeys of this size.

Birds were necropsied, and worm enumeration was performed on 8 and 16 birds for each

treatment at weeks 7 and 9 p.i., respectively.

2.3 Parasite Isolates

Eggs from a resistant (Sn 3.1F2F) and a susceptible (Ow 3.0) isolate of *A. dissimilis* were

obtained from passage of isolates whose drug susceptibility phenotypes were previously

confirmed (Collins, Jordan et al. 2019). Briefly, feces containing *A. dissimilis* eggs were washed

through a series of sieves, and then eggs were isolated by flotation using a solution with specific

gravity of 1.15 and centrifuged at 433g for 7 mins. The supernatant was collected on a 32um

mesh sieve and rinsed to remove flotation solution from eggs. Eggs were then stored in a tissue

culture flask containing water and 0.5% formalin and stored at 25°C to allow development to the

third stage larvae or infective stage.

2.4 *Infection and Treatment*

137  Starting on Day 0, 16 groups were infected with eggs of the resistant Sn 3.1F2F isolate

138 (hereafter referred to as Sn) and 16 groups were infected with the susceptible Ow 3.0 isolate

139 (hereafter referred to as Ow). Half of the groups infected with each isolate were then left

140 untreated and half received treatment with fenbendazole at weeks 4 and 8 (p.i.). In addition, 4

141 groups of 12 birds each were included as uninfected environmental sentinels.

142  Each week, fully larvated infective *A. dissimilis* eggs were mixed into feed at a target

143 inoculum dose of 25 eggs per bird. 3600 fully developed infective eggs in a volume of 1 ml were

144 pipetted onto 360 grams of feed, and the feed was then mixed well to disperse the eggs. Twenty

145 gr aliquots of the egg-contaminated feed containing approximately 300 eggs were then delivered

146 to each group each week by sprinkling on top of the fresh feed, adjusting to 250 and 225 total

147 eggs as birds were culled at weeks 7 and 8 p.i.

148  At weeks 4 and 8, treated groups were administered fenbendazole for five consecutive

149 days at a dosage of 1.25 mg/kg, which is 25% higher than the recommended label dose of 1.0

150 mg/kg. This higher dose was provided to maximize the likelihood that all birds consumed the

151 minimum full label dose. Treatment was administered using carboys delivering water to two side

152 by side pens. Dosage was calculated based on the total bird weight for both pens, selected 1 day

153 prior to the initiation of treatment. In order to maximize the likelihood that all birds would

154 consume the full dosage, the fenbendazole was administered in 90% of the estimated volume of

155 total daily water consumption. On all treatment days, the full volume of water containing the

156 fenbendazole was consumed.

157 *2.5 Statistical Analysis*

158  Statistical analyses were performed on weight gain and FCR values to model and identify

159 the effect of treatment, specifically, comparing turkeys infected with Ow and Sn, respectively.

Data from both week 4 and week 5 was considered as baseline in separate analyses. To account

for the growth across time, both linear and quadratic effects were introduced into the model.

Likelihood based methods were used for statistical analyses.

Specifically, the fitted model for Weight gain data was:

Log (Weight gain) for a bird at a time = log (baseline Weight gain) bird + b_1(time effect)

+b_2(time effect) ^2+ treatment effect +bird effect + error.

Conversely for FCR data was:

Log (FCR) for a bird at a time = log (baseline FCR) bird + b_1(time effect) +b_2(time effect)

^2+ treatment effect +bird effect + error.

The error was assumed to be normally distributed with mean 0 and variance that changed

with the treatment group. The errors between time points were modeled as an autoregressive

model of order 1 that changed across treatment groups. The bird effect was treated as a random

effect that was normally distributed with mean zero and independent of the error. All models

were selected using the Bayesian Information Criterion after considering several polynomial

models for time and different covariance structures. The normality of the error distributions was

evaluated using Shapiro-Wilks test.

The number of immature and adult worms recovered on day seven and day nine was

statistically analyzed, separately, using negative binomial regression with the logarithmic link

function. This model was chosen based on the likelihood criterion. In the analyses for adult

worms, data for the treated Ow group was not used in the analysis since all the observations were

zero. The model included the treatment group as an effect. All statistical comparisons were

evaluated at a 5% level of significance.

3.RESULTS

Analyses for weight gain and feed conversion ratio were performed separately using either week 4 or week 5 as baseline, with both analyses yielding consistent results. Week 5 was selected as the baseline for the results presented here, and results using week 4 as baseline are provided in Supplementary Tables 1 and 2.

Weight Gain. Based on the fitted model, the distribution of the errors was found to be normal (p-value=0.0871), and baseline was not a significant factor (p-value=0.3843). The slope for week was estimated to be -0.1154 (Std. Error=0.0810), and the slope for the square of time was estimated to be 0.0238 (Std. Error=0.0160), both of which were not significantly different from zero (p-values=0.1406, 0.1574). Weight gains (Table 1) were not significantly different between experimental groups (p-value=0.1283).

Feed Conversion Ratio. Based on the fitted model, the distribution of the errors was found to be normal (p-value=0.5040), and baseline was not a significant factor (p-value=0.6035). The slope for week was estimated to be 0.3571 (Std. Error= 0.0866) and slope for the square of time was estimated to be -0.0412 (Std. Error= 0.0171), both of which were significantly different from zero (p-values <0.0001, = 0.0179). Feed Conversion Ratio values are shown in Table 2 and Figure 1. Least square mean values for Feed Conversion Ratio (Table 3) differed overall between the groups (p-value=0.0036), therefore pairwise treatment comparisons were performed (Table 4). Based on these results, there were significant differences (p-value=0.0030) between treated and untreated birds infected with the drug-susceptible isolate (Ow), and between treated birds infected with the susceptible (Ow) and resistant (Sn) isolates (p-value=0.0150). However, there were no significant differences (p-value=0.2600) between treated and untreated birds infected with the resistant isolate (Sn).

Worm Counts at Week 7. The treated Ow group had no adults recovered, thus no

analyses were performed for this group. No significant differences were seen between the treated

and untreated Sn groups (p-value=0.8138). Additionally, there were no significant differences in

adult worms between the untreated Ow group and the untreated Sn (p-value=0.4832) or between

the untreated Ow group and treated Sn groups (p-value=0.2652). There were significant

differences between the untreated and treated groups in the number of immature worms

recovered for both the Ow (p-value = 0.0112) and Sn groups (p-value =0.0204). However, there

were no significant differences between the treated Ow and treated Sn groups in the number of

immature worms recovered (p-value = 0.1452). Mean worm counts for each treatment group at

Week 7 are shown (Table 5).

Worm Counts at Week 9. Very few adult worms were recovered from any of the groups

and most birds had no adult worms. Accordingly, no significant differences in adult worms were

noted. There were, however, significant differences in the number of immature worms between

the untreated and treated groups for both Ow birds (p-value <0.0001) and Sn birds (p-value

<0.0001). Additionally, significant differences were observed in the number of recovered

immature worms between the treated Ow group and treated Sn group of birds (p-value <0.0001).

Mean worm counts for each treatment group at Week 9 are shown (Table 5).

4.DISCUSSION

To the best of our knowledge, here we report findings of the first study measuring the

effects of drug-resistant *A. dissimilis* infection on turkeys. By infecting groups of birds with

either a known fenbendazole-susceptible and known fenbendazole-resistant isolate, we were able

to determine, using a mixed model for comparisons, the level of production loss caused by drug-

resistant parasites which were not removed by treatment. This model allowed for comparisons

that accounted for the random variability of worm burdens, feed consumption, etc. For these

comparisons, results were analyzed using both week 4 and week 5 as a baseline and no

differences in statistical results were seen using either week as baseline. Thus, we used week 5 as

baseline for all comparisons, as this was the point from which measurements would begin to

diverge as a consequence of failed treatments due to the presence of resistant worms.

Significant differences seen in FCR between the treated and untreated drug-susceptible

Ow groups indicate that the *A. dissimilis* infections were impairing FCR, and successful removal

of the drug-susceptible worms by treatment led to higher feed efficiency. In contrast, treatment

of birds infected with the drug-resistant Sn isolate did not yield an improvement in FCR.

Interestingly, no differences were seen in weight gain between groups, highlighting that this

effect on FCR is solely on feed consumption. Feed conversion efficiency is significantly

diminished, but birds appear to have gorged themselves on feed, making up for any possible

weight loss and driving FCR higher. Beginning in week 6 through the end of the study, the

treated Ow groups consumed an average of 230 grams less feed per week per bird as compared

to the treated Sn groups.

If the levels of production loss seen in this study due to the drug-resistant worms were

extended to the level of a house of 10,000 birds, this difference in feed usage would translate to

an extra 2.3 metric tons of feed needed per week. Using our feed cost of approximately $275 US

dollars/metric ton, this amounts to around $635 in extra feed costs/per week. Our grow-out only

lasted for 9 weeks, thus projections for a full grow-out if 16 weeks need to be made cautiously.

However, if this difference is projected onto a full 16 week grow-out, starting from week 5, total

extra feed costs due to effects of *A. dissimilis* on FCR for a 10,000-bird house would be

approximately $6,985.

The rather large differences recorded in FCR in this study are even more dramatic when

viewed in light of the low worm burdens achieved in this study. In a previous study with *A.*

dissimilis performed in commercial houses, mean worm burden from natural infections at day 56

post-infection was 13 adult worms per bird (Yazwinski, Rosenstein et al. 1993). In our recent

study, mean worm burdens from a bolus infection administered by gavage averaged 18.3 adult

worms per bird (Collins, Jordan et al. 2019). In contrast, at week 7 in our

current study (49 days post-infection), our untreated groups had average adult worm burdens per

bird of only 8.5 and 7.9 for Sn and Ow, respectively. This is only around 25% of what was seen

in the Yazwinski study at a similar time point, and around 44% of the burden seen in our

previous study. An estimated 200 total eggs per bird were given both in our previous, as well as

the current study. In the present study, our infection protocol was designed to replicate the trickle

infection birds would be expected to experience in a commercial house, however it failed to

produce the worm burdens seen in these previous studies. Despite this, we were still able to

determine the effects of treatment of worm burden in our treatment groups.

At week 7, in agreement with the significantly improved FCR, no adult parasites were

recovered from necropsy of Ow-Treated birds, indicating the high efficacy of fenbendazole

against this susceptible isolate by eliminating 100% of the adult burden. The few immature

parasites recovered from this group are most likely due to reinfection in the intervening post

treatment period. At this same time point, there were no significant differences in worm burdens

between treated and untreated Sn groups, and both had significantly higher adult worm burdens

than the treated Ow group, but not the Ow untreated group indicating the inability of treatment to

control parasites of the resistant isolate. This lack of control is in agreement with the lack of

improvement seen in FCR at this time point.

Although we were able to detect an impact on feed conversion, larger worm burdens

more typical of natural infections are needed to determine the full scale of drug-resistant worms

on FCR. It seems likely that higher worm burdens would have produced even greater negative

impacts on FCR than what are reported here. In addition to burdens, rearing time likely also

plays an important role in the effects on FCR. Longer grow out times with continual reinfection

due to environmental contamination with infective eggs, may lead to heavier burdens and

therefore increase the impacts. Due to limitations of our research space, which was designed for

chickens, it was necessary to prematurely terminate the study after 9 weeks. This contrasts to the

typical commercial grow out of 16-20 weeks. With a longer grow out period, it is possible that

the effects on FCR would continue or worsen causing further costs associated with resistant

parasites. Little is known about the population dynamics of *A. dissimilis*, and these dynamics,

would likely play a large role in determining the effects of resistant parasites in a full grow-out.

Additional studies will be needed to address this issue.

Overall, our data suggests that fenbendazole-resistant *A. dissimilis* have the potential to

impart substantial economic losses in the production of commercial turkeys. Presently, the

prevalence of resistance to fenbendazole is unknown, but may be much higher than is currently

realized (Collins, Jordan et al. 2019). Taken together, the results of our two recent studies

highlight the need for surveillance of resistance in helminths of poultry, for developing strategies

to prevent the development of drug resistance, and for developing strategies to address the

presence of drug resistant worms on a farm. Additional studies that better replicate the grow-out

time and worm infection levels that are typical on commercial turkey farms are needed to gain a

more accurate and full measure of the economic impacts of resistant *Ascaridia dissimilis* on

turkey production.

5. CONCLUSION

This study highlights the fact that *A. dissimilis* can significantly impact the economy of turkey

production even with low sub-clinical levels of infection. Thus, drug-resistant *A. dissimilis* have

the potential to significantly impact the production economy of turkeys.

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ETHICAL STATEMENT

All birds were handled under protocols approved by the University of Georgia Institutional

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Table 1. Weight Gain (kgs) for each treatment group by week. There were no significant

differences in weight gain between the groups.

	Treatment			
Week	**Ow-Treated**	**Ow-Untreated**	**Sn-Treated**	**Sn-Untreated**
1	0.14	0.15	0.16	0.16
2	0.20	0.21	0.17	0.21
3	0.33	0.36	0.34	0.35
4	0.43	0.45	0.45	0.44
5	0.44	0.54	0.48	0.49
6	0.70	0.63	0.71	0.65
7	0.77	0.79	0.80	0.83
8	0.56	0.70	0.61	0.64
9	0.72	0.77	0.89	0.76

377   **Table 2. Feed conversion ratio for each group by week. Feed conversion was calculated as**

378   **kilograms of feed divided by weight gain.**

		Treatment		
Week	Ow-Treated	Ow-Untreated	Sn-Treated	Sn-Untreated
1	1.26	1.21	1.16	1.18
2	1.30	1.34	1.82	1.32
3	1.50	1.46	1.40	1.48
4	1.55	1.56	1.52	1.64
5	2.04	1.77	1.87	2.01
6	1.71	2.01	1.83	1.94
7	1.74	2.12	2.05	2.01
8	2.59	2.90	2.98	3.16
9	2.48	2.83	2.58	2.82

390   **Figure 1. FCR for each treatment group over time.**

392   **Table 3. Least square means for FCR of each treatment group.**

Treatment	Estimate	Standard Error
Ow-Treated	0.7241	0.02995
Ow-Untreated	0.8645	0.02917
Sn-Treated	0.831	0.02762
Sn-Untreated	0.8755	0.02663

394   **Table 4. Pairwise comparisons for differences in least square means for FCR.**

Comparison	Estimate	Standard Error	Pr >|t|
Ow-Untreated vs. Ow- Treated	0.1404	0.04311	0.003
Ow-Treated vs. Sn-Treated	-0.1069	0.04113	0.015
Sn-Treated vs. Sn-Untreated	0.04452	0.03869	0.26

395 **Table 5. Mean worm counts by group at Week 7 and Week 9. For each treatment group, 8**

396 **birds were necropsied at week 7, and 16 birds were necropsied at week 9. Statistically**

397 **significant groups are designated. No analysis was done on total worm burdens.**

Group	Week 7			Week 9		
	Immature	Adults	Total	Immature	Adults	Total
Ow-Treated	1.88[c]	0.00[b]	1.88	0.13[b]	0.00[b]	0.13
Ow-Untreated	4.50[ab]	3.38[a]	7.88	10.94[a]	0.38[a]	11.31
Sn-Treated	3.13[bc]	2.25[a]	5.38	8.38[a]	1.06[a]	9.44
Sn-Untreated	6.00[a]	2.50[a]	8.50	11.13[a]	0.44[a]	11.56

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