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Genetic variability of resistance to *Eimeria acervulina* and *E. tenella* in chickens

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Summary. The genetic variability of 18 sire families of the Athens-Canadian randombred population infected with coccidiosis was assessed by examining the response variables of weight gain, packed red blood cell volume, mortality and coccidial lesions. A significant gain and PCV depression and high lesion scores for *Eimeria tenella* and *E. acervulina* were produced in the infected group compared to the noninfected group. Significant variation among the sire families was observed for all of the response variables except *E. acervulina* lesions and a significant sex x sire interaction was observed for weight gain. The heritability (h^2) estimates for the response variables revealed that resistance to coccidiosis in chickens is moderately heritable. The h^2 estimates for gain and PCV increased with the coccidial infections indicating that maximum progress in selecting for resistance should be made when the population was exposed to coccidial infection. Gain was positively correlated to the other measures of resistance and thus selecting for coccidial resistance should not reduce growth rates. PCV was similarly correlated but had higher positive correlation with *E. tenella* lesion. Percent mortality which is the selection parameter in most coccidial selection programs was correlated with resistance to coccidiosis. The phenotypic and genotypic correlations demonstrated that chickens susceptible to *E. tenella* were also susceptible to *E. acervulina*. Total lesion scores were moderately to highly correlated with the other variables and would be a suitable variable to use in coccidiosis experimentation including a genetic selection program for resistance. This study shows that progress could be made in selecting for resistance to coccidiosis in chickens using one or a combination of these response variables.

Key words: Coccidiosis – *Eimeria tenella* – *Eimeria acervulina* – Heritability – Genetic resistance

Introduction

Nine species of *Eimeria* (protozoa, coccidia) are known to infect chickens, of which 4 or 5 are important causes of clinical coccidiosis. These different intestinal parasite species vary in site of infection, their pathogenicity and lesions produced (Long and Reid 1982). For instance, *Eimeria acervulina* infects and produces lesions in the duodenal area and can cause growth depression and poor feed conversion, but does not cause sufficient hemorrhage to decrease packed red cell volume or deaths. In contrast, *E. tenella* infects the ceca and cause growth depression, hemorrhage, and often death. These two species are commonly encountered in poultry and cause tremendous economic problems.

Reduction of these economic losses due to coccidiosis would be of practical significance to poultrymen. Control of coccidiosis in poultry is primarily achieved by use of anticoccidial drugs and costs the U.S. broiler producers an estimated 70 million dollars/year (McDougald and Reid 1983). An alternative approach which has not been exploited is selection for genetic resistance to coccidiosis. Selective breeding for resistance to coccidiosis was suggested as early as 1927 by Johnson and by Rosenberg (1941), who demonstrated the feasibility of selecting for coccidial resistance. Rosenberg found significant differences in survivability between five different breeds of chickens and was able to select resistant and susceptible lines of chickens from one original population. Selective breeding programs for coccidial resistance have been conducted by Klimes and Orel (1969); Long (1968); Patterson et al. (1961); Rosenberg et al. (1954); and Champion (1951a, 1954). In these experiments survivability was used as the selection criterion. Survivability is certainly of importance in assessing the effects of cecal coccidiosis, but is a poor criterion for genetic selection. Mortality is discontinuous variation while the effects of the infection on the population are continuous, and it is difficult to manipulate the infections to a suitable range of mortality for maximum expression of genetic variability.

In the present experiments we examined some physiological parameters known to be associated with coccidiosis which might be used in selection for coccidi-

al resistance in chickens and the association between these parameters. Heritability estimates, phenotypic and genotypic correlations were determined for lesion scores, packed cell volume, percent mortality and weight gain in chickens infected with coccidiosis.

Materials and methods

A total of 842 chicks from 27 sire familes of the Athens Canadian random-bred population (Hess 1952) were hatched. The pedigreed chicks from each family were sexed, wingbanded and each sire family divided equally by sex into infected and noninfected groups. The sire families within each group were then mixed and placed in 5 floorpens (1.3 m × 2.4 m) with new litter and fed the standard University of Georgia broiler starter mash.

Two species of coccidia were used in this study. *Eimeria acervulina* specifically infects the epithelial cells of the duodenal loop causing the formation of whitish elongated lesions on the mucosal or serosal surfaces. Lesion score for an infection is based heavily on concentration of these ladderlike lesions. *Eimeria tenella* causes distinctive hemorrhagic lesions in the ceca. The integrity of the cecal wall and the amount of hemorrhaging and cecal core formation can be used to determine the lesion score for an *E. tenella* infection. Mortality and weight gain are also used to generally assess the response to the combined effects of the infection of *E. tenella* and *E. acervulina*, packed cell volume (PCV) and cecal lesion scores are more specific to *E. tenella* while duodenal lesion scores are specific for *E. acervulina*.

Infection with coccidia was prevented up to 21 days of age by feeding a strong anticoccidial drug. The drug feeding was discontinued for 3 days before start of the test. At 24 days of age all chicks were weighed, then half of the birds of each sex of each sire family were orally inoculated with 100,000 oocysts per bird using a combination of *Eimeria tenella* and *E. acervulina* by the methods described by Long et al. (1976). Five days post-inoculation blood samples were obtained from all chickens for PCV determination by the microhematocrit method (Johnson 1955). Seven days post-inoculation all birds were weighed and the infected chickens killed for lesion score (Johnson and Reid 1970). The scoring scale for the intestinal lesions caused by coccidial infections range from 0 (no lesions) to +4 (severe gross pathologic change).

Response to the infection was assessed both on the basis of within infected group variation and as a deviation of infected individuals from the mean of their non-infected sire family. The deviation was determined to examine variation due to coccidiosis separated from normal family variation.

The data from nine familes were discarded because of the small numbers in the sire family subclass (fewer than 10). The bird numbers in the remaining subclasses ranged from 11 to 35. The data were analyzed using either the Statistical Analysis System (SAS 79.3 B) (Barr et al. 1976) or General Linear Models procedure specifying the random option for obtaining expected mean squares. Heritabilities and genetic correlations within infected and noninfected groups were calculated based on half-sib analysis using the sire components of variance of the following model:

$$Y_{ijk} = u + a_i + b_j + c_{ij} + e_{ijk}$$

where u is the common mean, a_i is the effect of the i^{th} sire and is random; b_j is the effect of the j^{th} sex and is fixed; c_{ij} is the random interaction of the i^{th} sire and j^{th} sex; e_{eijk} is individual randomization within group. Genetic correlations were calculated for within infected groups as described by Yamada (1962) and Benyshek (1979) using a two-way classification model.

In order to obtain information based on the variability occurring because of the coccidiosis and not just natural variation, deviations of infected individuals from non-infected groups were determined. The deviations were obtained by subtracting the measurements of infected individuals from the mean of their non-infected control of the same sire family. Deviations were determined for initial and final weight, gain and PCV. The data set produced by these deviations was analyzed by the same model as stated before for differences between sire families, h^2, and correlations.

Results

Initial variation in body weight among the groups before infection was minimal. The average initial weight of the control was 159 g and for the infected group was 161 g. All of the 18 families were susceptible to coccidial infection, as shown by weight gain, PCV, percent mortality and *E. tenella* and *E. acervulina* lesions (Table 1). Average weight gain and PCV for all families were reduced 84% or 15%, respectively, by the two infections. When depression of gain and PCV were expressed as a deviation of infection from control, the infection reduced weight by 61 g and PCV by 5.9%.

Significant variation between sire families was observed for all the main effects except percent mortality and *E. acervulina* lesions. Initial and final weights varied significantly due to the sex of the chickens. The coccidial infection greatly suppressed the body weight gain and this probably eliminated a sex effect with this parameter. No sex difference was observed for PCV or coccidial lesions.

Heritability estimates

Heritability estimates were higher for all the main effects in the infected group than in the control group for the sexes combined and separated (Table 2) for weight gain, PCV and total lesion score. Estimates of actual variances and covariances for sire (sire component) and within family are also included in Table 2. Heritability estimates with fixed sex effects for body weight gain and PCV were increased from 0.17 and 0.15 for the controls to 0.20 and 0.30, respectively, for the infected group. These increases in h^2 indicate the appropriateness of using a coccidiosis challenge infection to allow for more expression of genetic variation.

Initial (IW) and final (FW) weights were highly heritable for both groups. The h^2 corrected for sex effect for weight gain and PCV were moderately high and moderate to low for percent mortality and lesion scores in the infected chickens.

Table 1. Variation between sire families in the effect of *E. acervulina* and *E. tenella* infection

Sire family	Number		24–31 day gain			PCV at 31 day			% Mortality	Lesion scores		
	Control	Infected	Control	Infected	Deviation	Control	Infected	Deviation		*E. acervulina*	*E. tenella*	Total
7	13	13	72	7	64	33	25	8	0.33	3.0	3.3	6.3
10	16	16	62	13	50	34	28	6	0.18	3.3	2.7	6.1
12	20	20	67	6	60	32	31	1	0.05	2.6	2.7	5.3
14	23	23	73	−4	77	32	26	5	0.21	2.9	2.6	5.5
15	24	24	68	13	55	34	13	21	0.18	2.8	2.4	5.3
16	24	24	65	12	53	33	23	9	0.17	2.9	2.8	5.8
18	18	18	69	7	62	34	28	6	0.20	3.0	3.1	6.1
19	21	21	75	22	54	34	26	8	0.14	3.1	2.8	5.9
20	15	15	65	−8	73	33	31	2	0.50	3.6	3.3	7.0
21	28	28	73	−2	75	33	29	4	0.29	3.2	3.3	6.5
23	30	30	74	6	68	32	28	4	0.26	3.1	2.8	6.0
24	31	31	74	7	67	32	27	4	0.06	3.1	2.9	6.0
25	35	35	80	24	55	32	31	2	0.13	3.2	2.6	5.8
26	21	21	69	17	52	32	27	6	0.28	2.8	3.4	6.3
27	31	31	62	15	47	32	26	6	0.19	2.9	2.9	5.8
28	22	22	70	14	57	31	28	4	0.16	3.0	2.6	5.7
29	16	16	80	16	63	33	31	2	0.16	3.1	2.8	5.9
30	11	11	69	6	63	32	24	9	0.28	3.3	3.3	6.7
$\bar{X}$ Male	194	194	Control	75.3±18.6		32.2±3.0						
			Infected	12.1± 6.1		28.2±6.1			19.5±40.5	3.1±0.78	2.9±0.98	6.0±1.3
			Deviation	−64.4±22.6		3.9±6.1						
$\bar{X}$ Female	205	250	Control	66.4±16.1		33.2±3.6						
			Infected	9.3±30.7		27.1±6.3			18.8±38.9	3.0±0.79	2.9±0.99	5.9±1.4
			Deviation	−56.7±30.9								
$\bar{X}$ M+F	399	399	Control	70.8±17.5		32.6±3.3						
			Infected	10.6±27.6		27.6±6.4			19.3±39.6	3.1±0.78	2.9±0.99	5.9±1.3
			Deviation	−60.0±28.3		−5.9±6.2						

Table 2. Heritability estimates and covariances corrected for sex effects for the noninfected and infected groups

	Initial weight		Final weight		Weight gain		σ^2_s	σ^2_w	PCV h^2		Covariance	
	h^2		h^2		h^2							
Group	Actual	Deviation	Actual	Deviation	Actual	Deviation			Actual	Deviation	σ^2_s	σ^2_w
Noninf.	0.74 ±0.32	–	0.66 ±0.32	–	0.17 ±0.11	–	16.4	136.7	0.15 ±0.10	–	0.44	11.31
Inf.	1.10 ±0.42	0.28 ±0.15	0.84 ±0.30	0.18 ±0.11	0.20 ±0.10	0.25 ±0.14	34.0	680.1	0.30 ±0.15	0.32 ±0.16	3.06	40.61

	Mortality %	*E. acervulina*	*E. tenella*	Total	σ^2_s	σ^2_w
	h^2	h^2	h^2	2		
Infected	0.10±0.15	0.08±0.05	0.18±0.11	0.16±0.11	0.072	1.79

$h^2 \pm SE$ = heritability ± standard error of the estimates

As a means of expressing h^2 of the main effects due to the influence of coccidiosis and not natural variation in gain and PCV, the heritability estimates for a corrected value, the deviation of control from infected, were determined. The h^2 for the deviation of IW and FW are lower than for IW and FW and the deviation of body weight gain and PCV are greater than gain and PCV. The lower h^2 for the deviation for IW and FW implies that even though these two parameters are highly heritable, their use as a measure of response to coccidial infection would only be moderately effective in selecting for coccidiosis resistance. The higher h^2 for the devi-

Table 3. The effect of *E. acervulina* and *E. tenella* infection on phenotypic and genotypic correlations with a random bred population of broiler chickens

Deviations	Initial weight				Final weight			
	Actual		Deviation		Actual		Deviation	
	r_p	r_g	r_p	r_g	r_p	r_g	r_p	r_g
Initial wt	0.91	0.63	–	–	–	–	–	–
Final wt	0.66	0.31	0.75	0.65	0.91	0.38	–	–
Weight gain	−0.05	0.33	−0.03	−0.35	0.55	−0.11	0.65	0.48
PCV	−0.05	0.56	−0.10	0.72	0.02	0.44	−0.03	0.25
Actual								
Final wt	0.80	0.96	0.71	0.51	0.91	0.38	–	–
Weight gain	0.03	0.29	−0.01	−0.15	0.62	0.53	0.64	0.39
PCV	−0.04	0.61	−0.10	0.68	0.03	0.60	−0.02	0.45
Mortality %	−0.02	–	0.01	–	−0.15	–	−0.13	–
E. acervulina	0.16	1.12	0.15	1.23	0.10	0.96	0.08	0.96
E. tenella	0.22	0.40	0.26	0.34	0.07	−0.14	0.10	0.04
Total lesions	0.26	0.46	0.28	0.56	0.11	0.25	0.12	0.41

Deviations	Weight gain				PCV			
	Actual		Deviation		Actual		Deviation	
	r_p	r_g	r_p	r_g	r_p	r_g	r_p	r_g
Gain	0.98	0.65	–	–	–	–	–	–
PCV	0.10	−0.04	0.07	−0.08	0.99	0.87	–	–
Actual PCV	0.11	0.21	0.08	−0.28	–	–	–	–
Mortality %	−0.21	–	−0.20	–	0.21	–	−0.21	–
E. acervulina	−0.04	−0.14	−0.05	0.57	0.03	0.08	0.01	−0.20
E. tenella	−0.16	−0.63	−0.15	−0.33	−0.28	−0.36	−0.27	−0.27
Total lesions	−0.14	−0.16	−0.15	−0.48	−0.19	−0.27	−0.20	−0.29

Actual	Mortality %[a]		*E. acervulina*[a]		*E. tenella*[a]		Total lesions[a]	
	r_p	r_g	r_p	r_g	r_p	r_g	r_p	r_g
E. acervulina	0.25	–	–	–	–	–	–	–
E. tenella	0.41	–	0.25	0.33	–	–	–	–
Total lesions	0.45	–	0.64	0.65	0.85	0.93	–	–

[a] Only actual values obtained

ation of gain and PCV suggest that their variation is due to the effects of coccidiosis and not natural family variation and would be good parameters to examine in a coccidial selection program. The use of PCV would of course be limited to *E tenella.*

Phenotypic and genotypic correlations

Phenotypic correlations were determined among all variables measured in the infected group (Table 3). There was a high phenotypic correlation between the determined deviations and the corresponding parameters. The weight parameters, initial weight (IW) and final weight (FW) generally had a low phenotypic correlation with the other parameters except a high correlation of final weight and gain. This is in agreement with the literature in that resistance to coccidiosis is not dependent on body weight at a specific point in time. Genotypically FW and gain were correlated with the other parameters; thus, genetic improvement could be made on a weight basis.

There was positive correlation of weight gain, phenotypically and genotypically, with FW and PCV and negative correlation with percent mortality and lesion scores. These correlations indicate that selection for coccidiosis resistance in this population should result in improved gain. PCV responded both phenotypically and genotypically in a similar way. The highest phenotypic correlation between PCV and another parameter

was with *E. tenella* lesions (– 0.28), and the lowest was with *E. acervulina* lesions (– 0.03). This difference is expected because of the hemorrhagic action of *E. tenella.*

In the studies using mortality as a selection parameter progress was made in selecting for *E. tenella* resistance. We also showed that percent mortality is correlated with resistance to coccidiosis. Percent mortality was low to moderately negatively correlated with initial and final weight, gain and PCV; and highly positively correlated with *E. tenella* and total lesions and moderate to *E. acervulina* lesions. Genotypically a 0.0 variance component correlation was calculated for all responses with mortality.

Both *E. acervulina* and *E. tenella* lesions were highly correlated with total lesion and were moderately correlated with each other suggesting that susceptibility to one *Eimeria* species is related to susceptibility to another *Eimeria* species and also that total lesion scores is a suitable parameter to assess response to coccidiosis.

Discussion

The heritability estimates for the response variables examined in this experiment reveal that resistance to coccidiosis is moderately heritable and selection progress could be made using one or a combination of these variables. The h^2 estimates for IW, FW, gain and PCV were the highest when calculated from the infected group when corrected for sex effects. The h^2 estimates for *E. tenella* lesions and total lesion scores were moderate to low and low for percent mortality and *E. acervulina* lesions. The use of coccidial infections could be recommended for a resistance selection program since the coccidial infections caused increased variation in the measured variables.

Jeffers (1969) reported a possible sex difference in resistance to *E. tenella* in two unselected chicken lines with a mean survival rate of 52% for females and 40% for males. We did not find a significant sex difference with any of the parameters except initial and final weight.

A significant genotypic and phenotypic relationship was noted between the pathogenic effects caused by the individual coccidial species. These positive correlations between *E. tenella* and *E. acervulina* indicate that a chicken susceptible to one species of *Eimeria* is probably susceptible to another species. A moderate to high correlation of total lesion scores with the other responses were noted and thus total lesion scores would be a suitable variable to use in coccidiosis experimentation.

These results further substantiate that selection for coccidial resistance is possible. This study indicates that selection for coccidial resistance must be conducted during a coccidiosis infection and that more variation in body weight gain is found with males than with females. Selection from the responses used in this study with more variation than mortality should allow significant progress in selection programs.

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