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Resistance Potential of Certain Breeds of Domestic Fowl Exposed to *Raillietina tetragona* Infections. V. Pathogenic Effects of the Cestode on Growing Chickens*

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Though several cestodes have been reported to be pathogenic to growing chickens, it appears that the harmful effects of *R. cesticillus* alone have been studied in detail (Ackert and Wisseman, 1946 ; Todd *et al.* 1950 ; Dutt, 1961 ; Nath and Pande, 1963 ; Mathur and Pande, 1969 and Botero and Reid, 1969). Relatively little is known about the pathogenicity of *Raillietina tetragona*, one of the wide-spread and most important of the fowl cestodes. The present study was, therefore, undertaken to elucidate by controlled experiments, the nature and extent of harmful effects, if any, produced by this cestode. To ascertain whether or not breed difference exists, four breeds of domestic fowl were used for this study.

Materials and Methods

Day-old White Leghorn, White Rock, Desi and Hybrid between White Leghorn and Desi were procured and maintained in the Laboratory on adequate diet. Twentyfour chickens, six of each breed, were used for the present study. Cysticercoids of *R. tetragona* were recovered from three species of ants, *Tetramorium simillimum*, *Tetramorium* sp. and *Pheidole* sp. When seven days old, three chickens of each breed were infected with 20 cysticercoids each by the method of Nadakal *et al.* (1970), and three chickens of the same age and weight from each breed were kept as controls (the difficulty of obtaining cysticercoids compelled us to limit the number of experimental and control birds). Both the infected and control chickens were kept individually in cages under parasite-free conditions. Weekly weight records of each chicken were made for a period of five weeks. Ten days after infection, the droppings of birds were examined daily and the prepatent period of worms determined. The number of proglottids discharged daily by the infected birds was recorded for one week before the birds were sacrificed. Elimination of strobilae or complete worms by host birds were also noted. The infected and control birds were sacrificed after five weeks. Before autopsy, hemoglobin contents of both infected and control birds were estimated by the acid hematin method and for correcting the amount of turbidity imparted by the nuclei of erythrocytes, the formula of Dukes and Schwarte (1931) was applied. For differential leucocyte counts, the copper peroxide method of Sato and Sekiya, modified by Nambiar *et al.* (1961) was adopted. The intestine of the birds were cut open and searched for complete worms, or scolices attached to the intestinal wall. The recovered worms were washed in normal saline and weight of each

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worm determined. Glycogen of host liver and worm was estimated quantitatively following the method of Seifter *et al.* (1950) using Spectronic-20 at the wave length of 620 mμ. Short pieces of intestine, with the attached scolices in the case of infected birds, liver and thymus gland tissues of both infected and control birds, were fixed in Bouin's fluid for histopathological studies. Serial sections, seven micra thick and stained with Heidenhain's iron hematoxylin and eosin, were prepared. Analysis of Variance method was employed in the statistical analysis. Differences between means were considered to be significant when P values were less than 0.05 and 0.01.

Results

The results of the present study are summarised in Tables 1 and 2. In the four breeds, no significant variation was noticed in the prepatent period. Maximum number of worms developed in White Rock and Desi (81.5%) and the minimum in Hybrids (40%). Despite higher percentage of worm development in the White Rock, the number of segments discharged per diem per worm was less then that of the three other breeds. But in the Hybrids with low percentage of worm development, the rate of segment discharge was almost equal to that of White Leghorn and Desi. The percentage of

Table 1 Data on host-parasite responses involving four breeds of chickens and the cestode *Raillietina tetragona*

Observations		White Leghorn	White Rock	Desi	Hybrid	P. Value
Prepatent period in days		12.33± 0.33	14.67± 0.33	13.00± 0.33	13.33± 0.44	>0.05
Number of worms developed		12.67± 2.33	16.33± 0.33	16.33± 0.75	8.00± 1.50	<0.05
Proglottid discharge per worm per diem		15.10± 1.09	12.70± 0.21	16.90± 0.52	15.90± 3.55	>0.05
Percentage of elimination of worms		12.04± 5.30	0	0	16.39± 1.13	<0.05
Length of worms in mm		108.03±26.89	158.13±28.71	124.73± 4.43	64.57±13.43	>0.05
Width of worms in mm		2.43± 0.23	2.80± 0.15	2.73± 0.29	3.37± 0.27	>0.05
Weight of worms in mg		89.9 ±27.53	122.67± 3.76	96.63±16.79	70.27±21.04	>0.05
Glycogen percentage in worms		3.89± 0.96	4.03± 0.05	3.47± 0.63	4.15± 0.61	>0.05
Liver glycogen	Experimental	1.40± 0.10	3.13± 0.81	2.43± 0.08	1.91± 0.21	>0.05
	Control	1.89± 0.16	2.65± 1.24	2.77± 0.10	1.69± 0.27	
Weight gain in grams	Experimental	281.67±28.59	247.33± 9.91	297.00±37.03	205.67± 2.18	<0.05
	Control	333.00±28.74	252.00± 6.24	267.33±40.70	219.00±23.03	
Hemoglobin gram percent	Experimental	8.27± 0.37	7.00± 0.65	9.17± 0.12	7.63± 0.34	<0.01
	Control	8.10± 0.91	6.97± 0.26	9.90± 0.10	8.47± 0.18	

Table 2 Differential leucocyte count of experimental and control chickens in percentage

Breed	Experimental					Control				
	*L	H	E	M	B	*L	H	E	M	B
White Leghorn	69	25	4	0	2	73	23	2	0	2
White Rock	65	27	6	1	1	71	21	4	2	2
Desi	72	24	2	2	0	73	21	2	1	3
Hybrid	71	22	6	0	1	64	31	4	1	0

* L, lymphocytes; H, heterophils; E, eosinophils; M, monocytes; B, basophils.

elimination was 12 (12.04±5.30) in White Leghorn and 16 (16.39±1.13) in Hybrid. In the other two breeds, there was no elimination. It was also noticed that worms gained maximum size and weight in White Rock and minimum in Hybrid. There was no appreciable difference in the glycogen content of worms developed in the four breeds. So also the liver glycogen content in the experimentals and controls of all the breeds showed no significant difference. There was about 15% reduction in weight in White Leghorn, 1% in White Rock and 6% in Hybrid, but 11.1% gain in Desi as compared with control birds. No difference in haemoglobin values could be noticed between experimentals and controls; however, breed difference in this respect was found significant at 1% level.

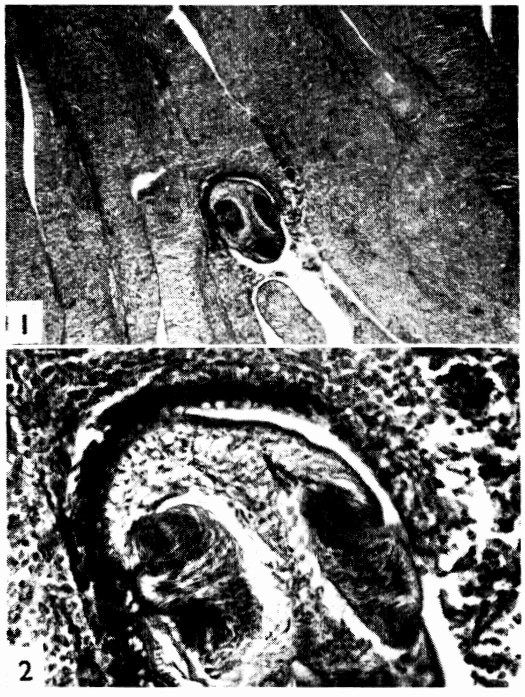
Study of serial sections of the intestine of the four breeds of fowl showed vacuolar degeneration and necrosis in the epithelium bordering the crypts of Lieberkuhn which were pronounced at the point of attachment of the parasites. A few mononuclear cells in the vicinity were also noticed and the connective tissue core of the villi showed increased fibroblastic elements and histiocytes. Focal degeneration and necrosis of the epithelium of the tip of the villi was also noticed. The epithelium of the neighbouring villi showed diffused early degenerative changes characterized by cytoplasmic vacuolation and karyopyknosis. There was moderate hypertrophy of the lymph nodes of the intestine and hyperplasia of the loose lymphoid tissue of the mucosa. In a few cases, the necrotic tips of the villi were covered by a moderate thick film of mucus mixed with exudates, in which a few erythrocytes were scattered. In one instance, moderate hypertrophy of the epithelial cells of the villi together with vacuolation giving the cells morphological resemblance to goblet cells was also observed (Figs. 1 and 2).

The thymus and liver tissues of the infected chickens showed no histopathological changes.

Discussion

The data presented above suggest that the four breeds of chickens vary considerably in their resistance-susceptibility relationship to *R. tetragona* infection. That breeds of chickens manifest variations in resistance to different helminths such as *Ascaridia lineata* (=*A. galli*), *Heterakis gallinae*, *Hymenolepis carioca* and *R. cesticillus* has been reported by Ackert *et al.* (1933, 1935), Todd and Culton (1949) and Todd *et al.* (1950) respectively. The general conclusion drawn by them was that heavy breeds were more resistant than the lighter breeds. Reid (1955), however, found no difference in resistance between the imported standard-bred and native Egyptian strains to *A. galli*.

On the basis of the percentage of development of worms, percentage of elimination of adult worms, and weight gain or loss of host birds, the four breeds can be broadly



Explanation of the Figures

Fig. 1 T. S of small intestine with attached *R. tetragona*. ×100

Fig. 2 T. S of small intestine showing the scolex of *R. tetragona* attached to the intestinal mucosa to show the histopathological changes. ×400

divided into two groups; one represented by White Leghorn and Hybrid and the other by White Rock and Desi. It appears that the former group is more resistant to *R. tetragona* infection than the latter as evidenced by lower percentage of worm development and expulsion of adult worms. However, the host birds suffered considerable weight loss. On the other hand, the latter group appeared to be more susceptible to infection, a fact substantiated by higher percentage of development of cysticercoids and absence of worm expulsion. The Desi fowl, however, differed from the White Rock in that there is considerable increase in the weight of the infected birds as against the controls. Though there was slight decrease in weight of the infected White Rock, this was insignificant when compared to that of other breeds.

Todd and Hansen (1951) reported that birds exposed to uniform parasitic infection weighed less if they had a few worms than if they had many and advanced the theory that the bird which expended energy in getting rid of worms gained less body weight than the one which tolerated the infection. Later Egerton and Hansen (1955) offered an alternative explanation for the weight loss of chickens infected with *A. galli*. According to them it is attributable to toxemia in the host, due to metabolic secretions or excretions of the worms entering the host's system. Reid and Carmon (1958), however, supported the classical view that damage caused by worms to the host is dependent upon the worm burden. In the present study we noted considerable weight loss in White Leghorn and Hybrid chickens which harbored fewer worms and eliminated a number of adult worms. Our findings thus seem to fit more with the interpretation of weight loss of birds in terms of expenditure of energy for worm expulsion. If we accept toxemia as a factor in the reduction of weight of host birds, White Rock and Desi breeds which harbored greater number of worms but eliminated none, should have sustained greater damage than the other breeds. Our observations also tend to differ from the view that host damage is proportional to worm burden.

The harmful effects of *R. tetragona* infection in White Rock was negligible as reflected in the weight loss of only 1%. The Desi fowls appeared to have possessed greater power of tolerance to infection, so that there was 11.1% increase in their weight. This may be partly attributable to their long association with the parasite resulting in a balanced host-parasite relationship of reciprocal advantage. However, in another series of experiments designed to study the effect of infection on egg-laying, administering 50 cysticercoids to each of the 5-month-old birds of the four breeds, we have obtained results indicating significant reduction in the rate of egg-production (data unpublished).

It seems that breed differences do not affect the prepatent period, proglottid discharge, size, weight and glycogen content of the worms. Depletion of liver glycogen has been observed in fowls infected with *A. galli*, whereas *Heterakis gallinarum* infection caused no change in it (Podgornova and Solov'eva, 1966). As in the latter case our data show that liver glycogen is unaffected, probably because the worms obtain their entire carbohydrate supply from the gut of the hosts. The difference in the haemoglobin gram percentage between experimental and control was not significant. However, breed difference in the haemoglobin gram percentage was noticed but it cannot be attributed entirely to parasitization. No eosinophilia was observed.

The present study has revealed that in all breeds of chickens the attachment of worms to the intestinal epithelium was only superficial. The histopathological changes brought about by the worms in the gut of the birds were of a mild type. Nath and Pande (1963), Srivastava and Pande (1967) and Chand (1970) also observed more or less similar histopathological changes in the birds infected with *R. tetragona*. However, the observed hyperplastic and hypertrophic changes in the lymph nodes of intestine, lymphoid tissues

of mucosa and epithelial cells of the villi seem to represent an incipient immunological response of the host birds. Liver and thymus gland tissues of the infected birds, we studied, showed no significant change differentiating them from those of the controls.

Within the limits of the data presented it is difficult to conclude whether or not *R. tetragona* is highly pathogenic to chickens. However, harmful effect of this cestode infection in White Leghorn and Hybrid birds is reflected in their weight loss. It appears that White Rock and Desi fowls are hardy birds capable of sustaining infection, not at the risk of significant weight loss but with definite advantage for the latter.

Summary

A study has been made of the pathogenic effects of *R. tetragona* infection in four breeds of domestic fowl, White Leghorn, White Rock, Desi and Hybrid (White Leghorn X Desi) maintained on normal diet. The White Leghorn and Hybrid birds suffered considerable weight loss; White Rock and Desi, while sustaining higher threshold of infection behaved differently in that the former lost weight by 1% and the latter gained by 11.1%. The histopathological changes in the intestine of infected birds were of a mild type. No significant difference in the haemoglobin values could be noticed between the experimental and control birds of all the four breeds; however breed difference in this respect was found significant at 1% level. There was no indication of eosinophilia due to infection.

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