

THE WILD TURKEY AS A HOST FOR *Heterakis gallinarum* AND *Histomonas meleagridis*

Authors: LUND, EVERETT E., CHUTE, ANNE M., and WILKINS, GARY C.

Source: Journal of Wildlife Diseases, 11(3) : 376-381

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-11.3.376>

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

THE WILD TURKEY AS A HOST FOR *Heterakis gallinarum* AND *Histomonas meleagridis*

EVERETT E. LUND, ANNE M. CHUTE and GARY C. WILKINS, Animal Parasitology Institute, Agricultural Research Services, United States Department of Agriculture, Beltsville, Maryland 20705, USA.

Abstract: Freshly embryonated eggs of *Heterakis gallinarum* gathered from naturally infected domestic turkeys and chickens developed the first 4 weeks essentially as well in young wild turkeys as in domestic poults, but then became progressively retarded and failed in most birds to result in females with fertile eggs. There was no significant difference in the prevalence or progress of infections with *Histomonas meleagridis* in the two kinds of turkeys, both of which differed from chickens only in that the latter had neither liver involvement nor mortality. In a second test, heterakids hatched from eggs stored 5-6 months at 4 C (comparable to overwintering) sustained very heavy losses in all birds, with greatly accelerated liberations of *H. meleagridis*. Few worms reached maturity and still fewer produced fertile eggs. In turkeys, and especially in wild turkeys, replacement of infective stages was so poor, that these birds were of no importance in contaminating the soil.

INTRODUCTION

Moore,¹⁰ in 1896, writing of "infectious entero-hepatitis" (= blackhead or histomoniasis) stated that "It is not known whether wild turkeys are affected." Nine years later, in a revision of Moore's bulletin, Mohler⁹ wrote: "It is now definitely known that wild turkeys, peacocks and also chickens are affected with this disease." Although early reports of the disease in the chicken and peafowl appear in the literature,^{1,2,8} the earliest specific reports of the disease in wild turkeys apparently remained unpublished, in the files of the Bureau of Animal Industry. Curtice³ tested the resistance of various breeds of turkeys to histomoniasis and reported as follows: "Inasmuch as wild, half-wild, Narragansett, Bronze, Mammoth Bronze, White Holland and mongrel, turkeys all entered into the experiments, and all suffered loss, no decided amount of immunity was possessed by them as shown even when the attempt was made to protect them from the disease." Schorger¹¹ cited many reports of

histomoniasis among pen-raised and free-ranging wild turkeys.

The experiments reported herein were designed to test the susceptibility of young wild turkeys (*Meleagris gallopavo*) to infections with *Histomonas meleagridis*, the protozoan that causes histomoniasis and *Heterakis gallinarum*, the cecal nematode that carries the histomonad. We also tested the potential of the wild turkey for contaminating the soil with infective stages of both parasites.

MATERIALS AND METHODS

Birds

The wild turkeys were purchased from a game hatchery as day-old poults. New Hampshire chickens and Beltsville Small White turkeys, used for comparison, were from flocks propagated for many years at the Institute. All birds were brooded 4 weeks on wire and then transferred to wire-floored cages. All chicks and poults were 5 weeks old when placed on experiment.

Parasites

The heterakid eggs used in each test were pooled from worms recovered from naturally infected New Hampshire chickens ranged with Beltsville Small White turkeys. Pretesting the freshly embryonated eggs showed that the population of *H. meleagridis* transmitted by these heterakids was composed mainly of strains only moderately virulent for young domestic turkeys. We estimated that the dose of approximately 125 embryonated eggs would not result in 100% incidence of infection with *H. meleagridis* in any breed of bird being tested, but might cause some mortality in domestic turkeys. A second test, conducted about 15 months after the first one, used heterakid eggs from the same source, but which had been kept at 4 C for 5-6 months after embryonation.

Procedure

The inocula were prepared by methods previously described.⁵

In Test 1, the heterakid eggs were used about 1 week after embryonation was complete. Forty-two wild turkey poults and the same number of domestic turkeys and chickens were each given approximately 125 embryonated heterakid eggs by pipette to the crop. An additional seven birds of each kind were kept as uninoculated controls. Six inoculated birds and one control bird of each kind were killed and examined at 10, 14, 17, 21, 28, 35 and 42 days after the infective feeding. Birds that died were examined as soon as possible after death. Each necropsy included a search for gross tissue responses to invasion by *H. meleagridis*, microscopic examination of cecal and liver lesions and cecal contents for the protozoan, and recovery of all heterakids. The worms were counted, separated according to sex, and measured, after which all mature females were placed in 0.5% formalin solution until their fertile eggs had embryonated. The average number of embryonated eggs per female was then determined by micro-

scopic examination of at least 10 individuals taken at random from each age group (35 and 42 days) and from each kind of host. Some of the female worms were fed intact, one per bird, to domestic turkeys. Eggs pooled from the females of the same sources were also fed to domestic turkeys in doses equal to the average number in the intact females that were fed. This method, previously described,⁶ enables one to determine approximately the ability of each kind of definitive host to contaminate the soil with *Histomonas*-bearing heterakid eggs. The reproductive potential (number of embryonated eggs produced per such egg given) of the heterakids in each kind of host was calculated as in an earlier study.⁷

The procedure for Test 2 was identical except that birds were necropsied at only six intervals, namely 10, 14, 17, 21, 35, and 42 days after inoculation.

RESULTS AND DISCUSSION

The results of the first test are summarized in Table 1 and those of the second test in Table 2, with all values presented in the same manner to facilitate comparison. All uninoculated control birds on both tests remained free of heterakids and histomonads.

The only noteworthy differences in response of the three kinds of birds in Test 1 to *H. meleagridis* were that the chickens had no liver involvement and none died. Only the most virulent strains of *H. meleagridis* cause liver lesions and mortality in New Hampshire chickens. All birds in which *H. meleagridis* was detected had gross cecal involvement, ranging from mild to severe in each kind of bird.

In Test 1, the overall recovery of *H. gallinarum* was satisfactory in the chickens, as was the recovery of mature heterakids, their length, the sex ratio, the production of embryonated eggs, and the reproductive potential. Under favorable circumstances, at 35 and 42 days, males tend to outnumber females about 1.3:1 (Lund, unpublished data), and the ratio among the heterakids from these

TABLE 1. Infections with *Histomonas meleagridis* and *Heterakis gallinarum* in birds each fed about 125 freshly embryonated heterakid eggs.

	Chickens	Turkeys	
		Domestic	Wild
No. of birds	42	42	38 ^①
<i>Histomonas meleagridis</i> :			
Prevalence of infection (%)	16.7 ^②	9.5	18.4
Avg. day of detection	15	17	19
Prevalence of gross liver lesions (%)	0	4.8	2.6
Mortality (%)	0	4.8	2.6
Avg. day of death	—	18	16
<i>Heterakis gallinarum</i> :			
% recovery (avg., all times)	46.0	26.7	23.2
Recovered at 35 and 42 days:			
Avg. no. per bird	48.8	38.4	19.6
Avg. no. males per bird	27	22.3	9.8
Avg. no. females per bird	21.8	16.1	9.8
Avg. no. embryonated eggs per female	62	72	57
Avg. no. embryonated eggs per bird ^③	1352	1159	559
Reproductive potential ^④	10.8	9.3	4.5
Avg. length at 42 days (mm):			
Males	9.5	8.9	8.8
Females	11.4	11.1	9.9
No. embryonated eggs per			
<i>Histomonas</i> infection in test poults:			
Fed as pooled eggs	—	375	250
Fed in intact females	—	—	385

① Four wild poults died early from causes unrelated to the experimental procedure.

② For chickens only, the prevalence of infection is based upon those necropsied 10-21 days after inoculation, because recovery occurs so promptly that evidences of infection may no longer be discernible at later observation periods.

③ Average no. embryonated eggs per female X avg. no. females per bird.

④ See Materials and Methods.

TABLE 2. Infections with *Histomonas meleagridis* and *Heterakis gallinarum* in birds each fed about 125 embryonated heterakid eggs stored 5½ months at 4 C.

	Chickens	Turkeys	
		Domestic	Wild
No. of birds	36	36	35 ^①
<i>Histomonas meleagridis</i> :			
Prevalence of infection (%)	54 ^②	42	49
Avg. day of detection	10	13	15
Prevalence of gross liver lesions (%)	0	2.8	0
Mortality (%)	0	5.6	0
Avg. day of death	—	19	—
<i>Heterakis gallinarum</i> :			
% recovery (avg., all times)	21.7	10.7	16.8
Recovered at 35 and 42 days:			
Avg. no. per bird	31.4	10.1	2.1
Avg. no. males per bird	15.7	5.2	0.9
Avg. no. females per bird	15.7	4.9	1.2
Avg. no. embryonated eggs per female	61	37	30
Avg. no. embryonated eggs per bird ^③	958	181	25
Reproductive potential ^④	7.7	1.4	0.2
Avg. length at 42 days (mm):			
Males	9.3	8.7	—
Females	10.8	10.3	—
No. embryonated eggs per			
<i>Histomonas</i> infection in test poults:			
Fed as pooled eggs	—	370	—
Fed in intact females	—	—	—

① Four wild poults died early from causes unrelated to the experimental procedure.

② See footnote 2, Table 1.

③ Avg. no. embryonated eggs per female X avg. no. females per bird.

④ See Materials and Methods.

chickens was 1.24:1. The reproductive potential of 10.8 is half again as high as the average for more than a dozen studies in which *H. gallinarum* associated with strains of *H. meleagridis* of moderate virulence was used.⁷ All of these circumstances indicate that the parasites used for Test 1 were well adapted to one of our comparison groups, the New Hampshire chickens.

H. meleagridis affected both domestic and wild turkey poults similarly. The overall recovery of heterakids was also similar for the two groups of turkeys, but only about half as high as that for the chickens. However, after the first 2 or 3 weeks, the survival and development of the worms in the wild turkey poults was poor. In domestic poults, where heterakids (once established) persisted with very small losses (only 4% between 10 days and maturity), the male: female ratio at maturity was 1.39:1, embryonation of eggs was excellent, and the reproductive potential was more than four times as great as our findings in past studies in which similar strains of parasites were used.⁷ In wild poults, losses of heterakids were 25% between 14 days and maturity. Losses of males were particularly heavy, and the sex ratio at maturity was 1:1. Only one 42-day female had any eggs that embryonated. The reproductive potential of 4.5 was due almost entirely to the performance of a few 35-day females. Mature heterakids were largest in chickens, intermediate in domestic poults, and smallest in wild poults.

Taken in their entirety, the results of Test 1 indicate that freshly embryonated eggs of a strain of *H. gallinarum* historically associated with chickens and domestic turkeys, and necessarily adapted to them, do not thrive as well in wild poults. As the final entry in Table 1 shows, the eggs that do embryonate are still capable of transmitting *H. meleagridis* at a near average rate.⁷

But unconfined wild turkeys do not always acquire their heterakids and histomonads as freshly embryonated eggs. Even among birds closely confined, acquisition may occur several months after

the heterakid eggs have embryonated, as in the spring, after the ground may have been frozen part of the time. Test 2, which differed from Test 1 primarily in the longer storage of the embryonated egg inoculum before use, provides data on the effects of aging on the heterakids.

In Test 2, the principal difference in response of the three kinds of birds to *H. meleagridis* was that mortality occurred only in the domestic turkey poults; two died and one had liver lesions. Inasmuch as a small percentage of the birds of any galliform species tested has little resistance to histomoniasis,⁷ the loss of two birds out of 36 (or out of 71, if all poults are considered) may have no other significance.

Overall recovery of *H. gallinarum* was poor for all groups in Test 2, and except in wild turkey poults, losses of worms were detectable by the 14th day. Inasmuch as histomonads are more frequently liberated by heterakids that die (especially as larvae) than by those that continue to develop to maturity,⁸ we consider the heavy loss of young heterakids to be largely responsible for the substantial increase in the incidence of infection with *H. meleagridis* in the chickens and poults in Test 2 as compared with those among these birds in Test 1. All results for heterakids indicate the severity of the struggle for survival. Mature worms of both sexes were smaller than those from the same kind of bird on Test 1. In no instance did the ratio of males to females at maturity vary much from 1:1, and only those females recovered from chickens produced embryonated eggs in numbers equalling those of birds in Test 1. The reproductive potential for heterakids in chickens was average, but for domestic poults was poor, even for worms transmitting *H. meleagridis* of moderate virulence.⁷ In wild turkey poults, the reproductive potential would not support even one more generation of worms. We can only conclude that the differences in the results of the two tests are due to the aging of the heterakid eggs used in Test 2. Larvae that have had to deplete the limited

energy resources within the egg during a long period of aging may not survive more than a few days in the new host, or may survive but be retarded in their development. We have long known⁴ that aging operates selectively much sooner on the heterakid than on the histomonad it may harbor.

Finally, our studies do not show appreciable differences between domestic and wild turkeys in their responses to *H. meleagridis*. Strains of *H. gallinarum* well adapted to domestic turkeys are not necessarily well adapted to wild turkeys, but as far as *H. gallinarum* is

concerned, aging of embryonated eggs is considerably more important than inherent physiologic differences in the cecal environment of the two kinds of turkeys. Closely confined wild turkeys, exposed to freshly embryonated heterakid eggs, could perpetuate the cecal worms (and histomonads that they might carry) to an extent sufficient to account for considerable losses. Unless they encounter strains of heterakids very different from the one we used, free-ranging wild turkey poults appear unlikely to contaminate soil with enough heterakid eggs to sustain populations of both parasites.

LITERATURE CITED

1. CHESTER, F. D. 1900. Common diseases of fowls. Their control and treatment. Del. Coll. Agr. Exp. Sta. Bull. 47.
2. CHESTER, F. D. and A. ROBIN. 1901. Enterohepatitis or blackhead of fowls. 12th Ann. Rep. Del. Agr. Exp. Sta., 60-66.
3. CURTICE, C. 1907. Further experiments in connection with the blackhead disease in turkeys. R.I. Agr. Exp. Sta. Bull. 124: 67-105.
4. LUND, E. E. 1960. Factors influencing the survival of *Heterakis* and *Histomonas* on soil. J. Parasit. 46 (Suppl.): 38.
5. LUND, E. E. and A. M. CHUTE. 1972. Transfer of ten-day *Heterakis gallinarum* larvae: effect on retention and development of the heterakids, and liberation of *Histomonas* and *Parahistomonas*. Exp. Parasit. 31: 361-369.
6. LUND, E. E. and A. M. CHUTE. 1972. Reciprocal responses of eight species of galliform birds and three parasites: *Heterakis gallinarum*, *Histomonas meleagridis*, and *Parahistomonas wenrichi*. J. Parasit. 58: 940-945.
7. LUND, E. E. and A. M. CHUTE. 1974. The reproductive potential of *Heterakis gallinarum* in various species of galliform birds: implications for survival of *H. gallinarum* and *Histomonas meleagridis* to modern times. Int. J. Parasit. 4: 455-461.
8. MELVIN, A. D. 1908. Twenty-third Annual Report of the Bureau of Animal Industry for the Year 1906. U.S. Dept. Agr., U.S. Gov. Printing Office, Washington, D.C.
9. MOHLER, J. R. 1905. Blackhead, or infectious entero-hepatitis, in turkeys. U.S. Dept. Agr. Circ. 5 (revised).
10. MOORE, V. A. 1896. The direct transmission of infectious entero-hepatitis in turkeys. U.S. Dept. Agr. Circ. 5.
11. SCHORGER, A. W. 1966. *The Wild Turkey: Its History and Domestication*. University of Oklahoma Press, Norman, Oklahoma.

Received for publication 17 December 1974