LEUCOCYTOZOONOSIS IN CANADA GEESE AT THE SENEY NATIONAL WILDLIFE REFUGE[1]

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Abstract: A history is given of the Seney National Wildlife Refuge and the losses of goslings of Canada geese (*Branta canadensis*) recorded since inception of the refuge in 1935. Since 1960, when more reliable data became available, losses have been extensive every 4 years. Gosling deaths are attributed to the infection with *Leucocytozoon simondi*. The blackfly (*Simulium innocens*) is considered to be the prime vector in the transmission of this blood parasite to goslings.

INTRODUCTION

The Canada goose, *Branta canadensis* (L.), is native to North America, breeding in the northern reaches of the United States and Canada and overwintering in the south. At least 10 subspecies are recognized in the latest checklist of the American Ornithologists' Union.[1] Efforts have been made to manage these birds by regulating their harvest and by establishing refuges for breeding and wintering populations.

The Seney National Wildlife Refuge was established in 1935 on the northern peninsula of Michigan, between Germfask and Seney. It encompasses 38,678 ha, including 24,682 ha of marsh and 2,932 ha of open water.[3,2] Most of the open water area is contained in 21 impoundments that were created by diking; these impoundments range from 11 to over 400 ha. Water levels are manipulated by controlling the supply delivered through diversion ditches from three streams; transfer ditches enable water to flow from one impoundment to another. The ditches provide excellent larval habitat for some species of blackflies (Simuliidae).

HISTORY OF GOSLING LOSSES

According to Crawford,[4] the region around Seney was the breeding ground for Canada geese until the summer of 1910 when their numbers rapidly diminished. The last geese were seen in Walsh Ditch (now part of the refuge) in 1929. No resident Canada geese inhabited the area until 332 pinioned birds were released into the new refuge in January, 1936. The birds were a gift from Henry M. Wallace,[5] who had

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[4] Crawford, E. E. 1936. Experimental Canada geese. Seney National Wildlife Refuge files.

[5] Johnson, C. S. 1944. Report in files of the Seney National Wildlife Refuge.

raised Canada geese since the early 1920's on a 2,400 ha farm near Highland, Michigan, about 64 km west of Detroit. Wallace's flock was started with four wild birds obtained from Owatonna, Minnesota.[4] [6] During the first breeding season at least five pairs of the newly stocked birds were successful in hatching a total of 19 goslings.[5,24] This marked the beginning of the Seney goose flock. All offspring of the pinioned flock at Seney were allowed to fly free, and they eventually established a migrating and homing pattern. Hanson[13] identified these birds as giant Canada geese, *Branta canadensis maxima.*

The Seney experiment was the first attempt to establish a major flock of breeding Canada geese on a national wildlife refuge. Records show that the flock increased yearly from the original 332 in 1936 to 3,000 in 1956. There was a poor gosling crop in 1939, followed by temporary setbacks in 1943-44 and again in 1951.

Johnson[5] reported that the 1942 population consisted of 422 birds, of which 199 were migrants that had returned to Seney to breed. He attributed the 1943 loss of many goslings in the last week of May and the first week of June to cold rainy weather at a time when the goslings were very young. He apparently recognized a population plateau and a slowing growth of the flock: "The Canada goose flock has passed through nonreproductiveness followed by various stages of growth and decimation." During most of these early years, the flock was disturbed as little as possible until the goslings were well along in the stage 2 age group (when the birds are about 45 days old and almost fully feathered) for fear the nests and young might be deserted by the parents. It was not clear in Johnson's reports whether nesting failures or brood failures were the cause of reduced production.

In 1945, dead and sick goslings were observed but very few field notes appear to have been made at that time; a Seney Refuge Quarterly Narrative Report estimated the goose population at 500-600 birds.[32] In 1949 a general increase in waterfowl in the Upper Peninsula was not reflected at the Seney Refuge. Reports for 1950 indicated a population drop from 600 to 500 birds.

By 1954 the population of Canada geese had grown to an estimated 2,700 with 250 breeding pairs producing 950 young, of which 750 survived to migration. According to Henry[7] "Our losses were heavier this year . . . . . We had a terrific hatch of blackflies about the time of the gosling hatch."

The gosling yield was estimated to be low in 1955, 750 compared to 950 in the previous year. In 1956, however, the yield was 1,000 and there were no reports of sick goslings. In 1957, both the number of nests and number of goslings per brood decreased. The decrease in nests was attributed to heavy hunting pressure during the previous fall. The greatest losses were in the broods that hatched late.

Production was poor again in 1960. The estimated hatch was 790, about 30% less than the previous year. Heavy rains with rapid spring thawing produced floods that were thought to have caused the lower hatch. William French,[8] refuge biologist, attributed the lower production to the "worst water conditions in 50 years in the Upper Peninsula of Michigan." But subsequent field checks showed little or no nest damage attributable to high water. During late June, over 50 gosling carcasses were recovered from areas that were above high water levels. On 5 September it was estimated that only 100 goslings remained, thus indicating a loss of approximately 690 goslings.

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[6] Sypulski, J. L. 1954. History of Seney Refuge geese, 1936-1942. Seney National Wildlife Refuge files.

[7] Henry, C. J. 1954. Report in files of the Seney National Wildlife Refuge.

[8] French, W. 1960. Memorandum in the files of the Seney National Wildlife Refuge.

Early techniques for estimating populations and production were not standardized and were not consistently reliable. Population estimates were as great as 3,000 in 1956. Critical review of these techniques and increased emphasis on more adequate methods of determining population size led Sherwood[9] to conclude that estimates of the population prior to 1960 were probably too high by 500 to 1,000 birds.

Gosling mortality in 1960 was alarmingly high. We have since learned that heavy losses may be expected once every 4 years. Since census methods were not reliable prior to 1960, cyclic losses could have been missed. The early records, nevertheless, indicated that noticeable setbacks in flock growth occurred several times prior to 1960.[32]

Gosling losses since 1960 are shown in Table 1. Improved counting methods and marking of individual birds with plastic neck bands identifiable at a distance provided a better base for censusing than in earlier years. Even the figures presented for recent years, however, should be considered to be estimates.

TABLE 1. Annual estimates of production and mortality of Canada goose goslings at the Seney National Wildlife Refuge.

Year	Goslings Hatched	Gosling Mortality	Percent Mortality
1960	790	690	87
1962	819	269	33
1963	609	134	22
1964	627	527	84
1965	676	186	28
1966	818	143	17
1967	912	150	16
1968	1022	730	71
1969	1091	545	50
1970	1181	381	32
1971	1035	335	32
1972	425	325	76

OCCURRENCE OF LEUCOCYTOZOON

The genus was first found in North America by Wickware[40] who reported it from a domestic duck in Ontario, Canada. He named the parasite *L. anatis*. O'Roke[31] observed *Leucocytozoon* in blood films from several species of wild ducks in northern Michigan and claimed that extensive losses occurred among ducklings. *Leucocytozoon* has been reported from a wide variety of birds in North America[16] and elsewhere[8] and from many species of Anatidae from North America.[20,21]

The first North American report of *Leucocytozoon* from a goose was from domestic bird in Quebec in 1933.[41] but several investigators have reported *L. anseris* as a pathogen of domestic geese in central Europe.[23,25,26,33] *Leucocytozoon* in a Canada goose was first reported from Cape Cod, Massachusetts;[15] it

[9] Sherwood, G. A. 1963. Quarterly Report in the files at the Seney National Wildlife Refuge.

has subsequently been reported in subspecies of *B. canadensis* from Michigan,[3,16] Illinois,[28] southern California,[42] Wisconsin,[36] Quebec,[27] Northwest Territories,[6] and Labrador.[4] The species of parasite in the Canada goose is considered to be *L. simondi*.[14] Several authors have mentioned the occurrence of *Leucocytozoon* at the Seney Refuge.[3,17,18,19,20,21,22,34,35,36,37,39] The following observation was recorded in a 1936 Seney Refuge Quarterly Narrative Report in the Seney Refuge files: "The young goslings were noted to have a heavy infestation of blackflies at the base of their necks . . . . Dr. E. C. O'Roke suggested that our geese might be susceptible to the prevalent malaria-like disease, *Leucocytozoon*, found amongst our native ducks." This suspicion was confirmed in 1945 when O'Roke diagnosed *Leucocytozoon* in a dead gosling from Seney. Beard[10] observed sick goslings in 1949 and sent blood smears from 38 goslings and two adult Canada geese from Seney to the senior author who reported very heavy parasitemias with *Leucocytozoon* in the goslings and suggested this parasite might have been the main contributory factor in the losses.

Since 1950 the Patuxent Wildlife Research Center has given increasing attention to the problem at the Seney Refuge in an effort to determine the cause of the losses of goslings. The high incidence of *L. simondi* incriminates this agent above all others. Although other parasites and possible disease-causing agents have been recorded, they have had a relatively low incidence compared with the *Leucocytozoon*. Other organisms observed include trypanosomes and coccidia, *Trichomonas* spp., *Haemoproteus* (*Parahaemoproteus*) *nettionis, Plasmodium* spp., *Tetrameres* sp., *Amidostomum anseris, Aspergillus* and viruses. None of these has been shown to be a cause of the epizootic losses. Autopsies of dead goslings revealed less than 5% with aspergillosis as a possible cause of death (one or two birds per year, some years none), and no pathological condition, other than that attributed to *Leucocytozoon*, was observed that was suggestive of an infectious agent.

Goslings have been examined in every year since 1950, except in 1956, and *L. simondi* has been consistently present. Some birds usually die during the first week in June; goslings 2 to 7 weeks of age appear to be the most vulnerable. At the time of death, blood smears show a high level of parasitemia with the round forms of *L. simondi*. Occasionally, particularly in the youngest age group, death occurs without parasitemia. In such birds we have demonstrated overwhelming infection in the tissues with schizogonic stages of the parasite, which suggests death from the parasitic infection at an early stage prior to appearance of parasites in the blood. Experimental infection, either by exposure of sentinel birds or by inoculation with infected vector flies or sporozoites obtained from such flies, indicates a prepatent period of 4 to 7 days, occasionally up to 2 weeks, before parasitemia can be demonstrated. Surviving birds lose their heavy parasitemia within a matter of weeks, the level of parasitemia subsiding to one parasite per 100,000 red blood cells or lower. With such low intensity of infection, parasites are observed only occasionally in routine microscopic examination. When infected birds are maintained in captivity, the parasitemia remains at this low level until the following spring; routine periodic blood films examined during this period rarely show an occasional elongate form. Parasitemia increases about the time of egg-laying and parasites can be readily demonstrated. Chernin[7] was the first to show that regulation of the day length under laboratory conditions could stimulate the female goose to lay eggs about a month early; under such treatment, both the male and female birds exhibited parasitemia a month earlier than under natural conditions. One of us[2] was able to demonstrate that an increase in parasi-

[10] Beard, E. 1949. Report in files of the Seney National Wildlife Refuge.

temia could also be produced by stress stimuli at any time following initial recovery.

In the early 1950's, our observations were limited to examination of a few sick or dead birds submitted to the laboratory for examination, and to observations made during brief visits to the Seney Refuge at the height of the losses. Dead and sick birds that were examined in 1954, which was a year of heavy losses, demonstrated a very high level of parasitemia (many parasites observable in each microscopic field—often over 50 parasites per 100 red blood cells). No losses were recorded in 1956; however, no birds were examined for evidence of *L. simondi* during that year.

Data from the studies during the 1950's led to the assumption that all goslings became infected with *Leucocytozoon*, and the more extensive data of recent years substantiated this assumption. Beginning with the summer of 1959, more frequent visits were made to the study site. As a result, we were able to determine that most goslings died between June 3 and June 10.

In 1963, and again in 1964, we observed the goose flock upon its arrival at Seney in the spring. Pairs chose their nesting territories and settled down to defend them against intrusion by the rest of the flock. Unattached birds, however, continued to show excitable and aggressive behavior. Following a suggestion by Barrow,[2] we attempted to correlate behavior with the intensity of infection. A number of birds were captured by means of a cannon net and banded with colored plastic neck bands which made field identification possible. Examination of blood smears indicated that the unattached birds had an intensity of parasitemia 2 or 3 times the level in the birds that had already chosen nest territories. Just prior to egg-laying time more than 80% of all the birds had a readily demonstrable parasitemia with *L. simondi*.

On the basis of the knowledge currently available, we conclude that *L. simondi* is the prime cause of the die-off in the Canada goose gosling population at the Seney National Wildlife Refuge.

We have been unable to explain the cyclic fluctuation in the losses; all evidence points to 100% infection in the goslings every year. We have unsuccessfully attempted to correlate weather conditions, hunting pressure, predation rate, and other parasites (which may provide an additive or synergistic effect) with the fluctuation in losses. Immune response may contribute to the cycles, for there is an indication that younger birds succumb more readily, but the data are not conclusive. Some investigators claim that the number of sporozoites in initial or repeated exposures may be a factor to consider.[11]

VECTORS OF LEUCOCYTOZOON

O'Roke[31] reported that *Leucocytozoon* of Anatidae is transmitted by simuliids. Studies in Ontario[9,11,12] demonstrated that the prime natural vectors of *L. simondi* in ducks are *Simulium rugglesi* and *S. (E.) anatinum*. Studies at the University of Michigan Biological Station confirmed *S. rugglesi* as a prime vector.[3] In 1960-1964 it was determined that *S. rugglesi* was the dominant blackfly in the Seney area from early June through mid-July and was capable of transmitting *L. simondi* to geese as well as to ducks.

Before 1964 there were few studies of the blackflies of the Seney area; only four species had been reported in the literature. Since that time, over 100 species, of which 35 are ornithophilic, have been identified in and around the Seney Refuge.[36] Specimens were collected by sweeping (netting), by using birds as bait, and by collecting eggs, larvae, and pupae from breeding habitats in water courses. Studies of the occurrence of these flies at Seney indicate that *S. rugglesi* is not present in substantial numbers until after the first week of June, by which time extensive deaths of goslings with heavy infections of *Leucocytozoon* have already occurred. It appears, therefore, that *S. rugglesi* is probably not involved initially in the transmission of *L. simondi* in the local goose population.

Of the species of Simuliidae prevalent in the region earlier in the season, we first considered *Cnephia invenusta* to be a potential natural vector,[37] but subsequent studies indicated that this was incorrect. Field and laboratory experiments demonstrated that *C. invenusta* is incapable of transmission.[35]

Although 35 ornithophilic species of blackflies were taken at Seney during our observations, only four were taken from birds (*C. invenusta, C. taeniatifrons, S. innocens* and *S. rugglesi*) in sufficient abundance to be suspect vectors. Of the potential vectors present during May, the prime suspect is *S. innocens.*[11] Both laboratory and field studies indicate that it is a potential candidate.[35] In some years, particularly 1972, it was the only ornithophilic species present just prior to and during the time of the seasonal epizootic. Though immature stages (larvae and pupae) of *S. euryadminiculum* have been taken at Seney, no adult flies have been taken from exposed ducks and geese. Further study of this species will be required to determine whether it may serve as a vector. In Algonquin Park it is restricted to feeding on loons and appears to be an unlikely natural vector.[5,29] Both *S. innocens* and *S. rugglesi* are able vectors.[35].

S. innocens is believed to be the prime vector in transmission of *Leucocytozoon* to the goslings. However, a high incidence of this parasite is recognized in *S. rugglesi* throughout June to mid-July, as at Algonquin Park in Ontario.[10] We, as well as many of our colleagues, have collected *S. rugglesi* in the northern peninsula of Michigan as late as the last week in July, taken them to the laboratory and successfully produced infected ducks and geese by inoculation of triturated flies. We have collected ducks which hatch later in the season, at both Seney Refuge and other areas of northern Michigan that demonstrate a parasitemia up to mid-September; it is more likely, therefore, that the heavy prevalence of *L. simondi* in these flies was obtained from ducks than from geese.

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