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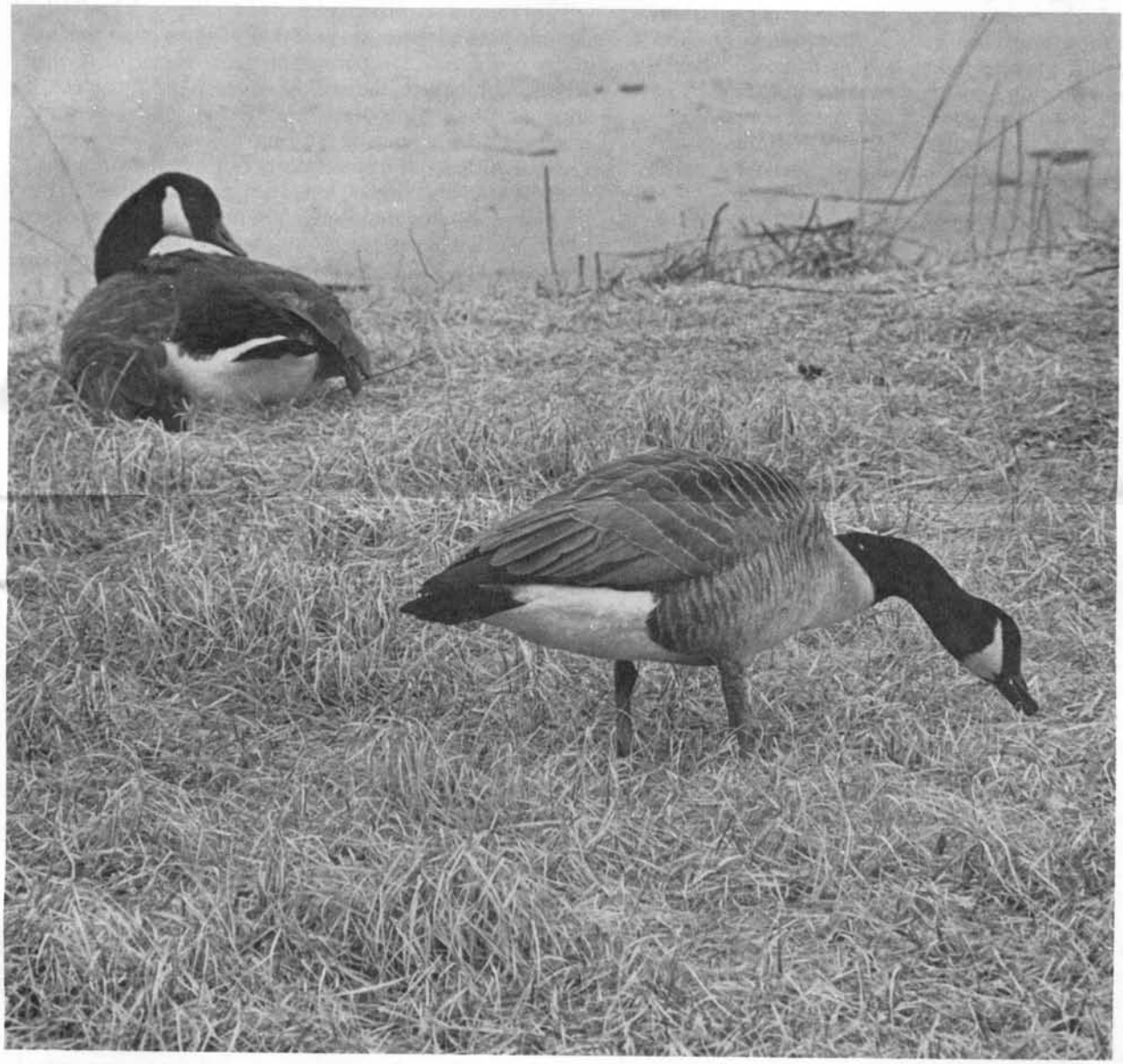

The Canada goose problem. See Page 2

THE CONNECTICUT AGRICULTURAL EXPERIMENT STATION NEW HAVEN

Manipulating feeding sites reduces damage caused by Canada geese

By Michael R. Conover

The Canada goose is one of the most beloved—and disliked—birds in Connecticut. For most people, it is a thing of beauty and a source of enjoyment. For farmers, however, it is a pest that devours or tramples their grain and green forage crops. In recent years, the Canada goose has also become a serious nuisance in some urban and suburban areas when large resident flocks feed on grass in parks, playing fields, golf courses, and backyards. Some playing fields and beaches in Connecticut have been closed temporarily, and some people have been afraid to let their children play in their own backyards because geese litter these sites with fecal material, which is unsightly, messy, and may be unhealthy.

These problems are not limited to Connecticut. A survey that Gregory Chasko and I conducted of golf course superintendents in the East revealed that geese were a problem in most states, especially in the coastal states from Massachusetts to Maryland. To determine the severity of the problem, we asked managers who complained about geese how much they would be willing to spend yearly to eliminate the problem; the average response was $444. If we extrapolate this amount to all golf courses in the East, managers would be willing to spend close to $1 million per year to control Canada geese. Since geese are also a nuisance in the West and in Canada and golf courses aren't the only places with goose problems, geese appear to be a multi-million dollar problem. Unfortunately, there is no easy solution to this nuisance. The problem is compounded by the fact that many of the geese no longer fly south for the winter; instead, they have taken up permanent residence in Connecticut.

Geese are protected by federal law; therefore, they may only be killed during the fall hunting season. The golf course mangers we surveyed had tried many nonlethal techniques to chase geese away. They tried to scare them with guns, firecrackers, predator models, balloons, dogs, noise makers, swan decoys, and models of such enemies as hawks and owls. No technique worked most of the time. In the past, federal workers sometimes rounded up geese during the summer molt when the birds could not fly and transported them to other areas. This, however, was time-consuming, could only be done once a year, and was at best temporary. Last year the round-up was suspended because of fear that it might spread bird diseases.

How could the geese be kept out of sensitive areas without harming the birds? I experimented to see if geese would avoid feeding on grass that was sprayed with a chemical repellent, methiocarb. This chemical makes birds slightly ill when they consume it but otherwise does not harm them. It already is used to keep birds from eating ripening blueberries and cherries.

Using caged Canada geese, I found that if given a choice they avoided feeding on turf sprayed with methiocarb at a rate of 3 pounds per acre. Geese given the opportunity to eat methiocarb-treated grass for two months suffered no noticeable ill-effects.

I next tested the effectiveness of methiocarb by studying one-acre plots on several golf courses experiencing goose problems. After counting goose feces for several weeks to measure the use of these plots by geese, I sprayed some plots with methiocarb and left some unsprayed as a control. During the next two weeks, geese avoided the treated plots but not the untreated ones. Within two weeks, however, geese returned to the treated plots because insufficient methiocarb remained to inhibit their feeding.

Since these experiments indicated that geese could be repelled by spraying grass with methiocarb, I conducted another experiment to see the effect of spraying an entire feeding site instead of part of one. I tested this by spraying entire yards around businesses and homes where geese were a problem. I found that geese avoided these sites for 6 to 10 weeks. Geese probably avoided these sites longer than the small sprayed plots on the golf courses because when only a small plot was sprayed, geese feeding nearby learned quickly when the treated turf was again safe to eat. When an entire feeding site was sprayed, however, geese moved to other feeding areas. Because the geese were somewhere else when the grass once again became palatable, it took them longer to find this out. These experiments are described in more detail in a report to be published in *The Journal of Wildlife Management*.

It may be possible to eliminate goose problems at some sites by planting grass species which geese find unpalatable. Consequently, I tested the feeding preferences of captive Canada geese for five different grasses: colonial bentgrass, Kentucky bluegrass, K-31 (a tall fescue variety), perennial ryegrass, and red fescue. The geese showed a strong preference for Kentucky bluegrass and a strong dislike for K-31. When no choice was available, however, hungry geese fed on any of the five grasses. This suggests that planting K-31 instead of Kentucky bluegrass at a problem site may discourage geese from feeding there as long as other grass or food is available elsewhere.

To determine why geese disliked K-31, I examined the physical and nutritional properties of the five grasses. I found that the feeding preferences did not relate to the amount of protein or carbohydrates in the grass, but instead they related to the force required to sever a given amount of leaves. This is a measure of how tough or tender the leaves are. Hence, my data indicate that the geese selected grass with tender leaves and avoided

grass with tough leaves.

Because geese bring enjoyment to many people, we might consider nuisance Canada geese the right birds in the wrong place. These experiments may help keep the geese in the right place where they may be enjoyed, rather than in the wrong place where they are a pest.

Witloof chicory, alias Belgian endive A future vegetable staple?

By David E. Hill

The root of chicory has long been known as a coffee substitute and its wild flowers as blue dots along roadsides in summer. One chicory, however, witloof (*Chicorium intybus*), is becoming increasingly known as a gourmet salad ingredient or a cooked vegetable. Its rise in popularity is demonstrated by imports from Belgium increasing from 440 tons in 1976 to over 3,000 tons in 1983. Under its popular name, "Belgian endive," witloof (pronounced wit-loaf) chicory is sold in many markets.

In 1984 I began to investigate witloof chicory as a new crop for Connecticut because of its value and increasing popularity, and because the climate and soils in Connecticut resemble the climate and soils where it is grown in Europe.

Cultivation of witloof chicory is complicated. The plants are grown for their roots, harvested in the fall, placed in cool storage, replanted and forced to sprout in a darkened enclosure during the winter.

Although several domestic suppliers offer seed of witloof chicory, I imported cultivars from Holland to provide forcing times from late fall through early spring and a variety of forcing conditions. Our trial crops were grown in a sandy loam at the Valley Laboratory in Windsor and on a moderately stony loam at Lockwood Farm in Mt. Carmel. Both soils were fertilized with 35 lbs/1000 ft^2 5-10-20 supplemented with 20 lbs/1000 ft^2 epsom salts. High concentrations of phosphorus, potassium and magnesium are essential to develop strong roots, and the low nitrogen content discourages top growth. Limestone was added to raise the pH to 6.5. The seed was sown in mid-May. The herbicide pronamide was applied after seeding and watered in to control weeds. The plants were thinned to 6 inches apart when they were about 3 inches high. Our experiments showed that a 15-inch row spacing produced 10-20% small, immature roots. An 18-inch row spacing would have reduced competition among plants and produced a higher proportion of large roots. As with most vegetables irrigation was necessary, especially in the sandy soil, to provide an inch of water per week.

Maturity was determined by splitting a root longitudinally and examining the white patch of tissue the size of a fingernail just below the vegetative crown. This tissue, from which the regrowth occurs during forcing, should be ¼-⅜ inch thick. Thicknesses less than ¼ inch indicate immaturity and thicknesses ⅜-½ inch indicate overmaturity. When forced, overmature roots produce undesired multiple heads or crown shoots. An overmature crop in the field will begin to bolt as the vegetative stem elongates, sending forth the familiar blue flowers of chicory. Plants that bolt cannot be forced. Our crop matured rapidly and was harvested in late-August. Bolting among the cultivars (10 to 30%) was caused by cool soil in the spring, which vernalized the biennial plants and reduced their life cycle to one year.

At maturity, the entire plants were dug and left on the

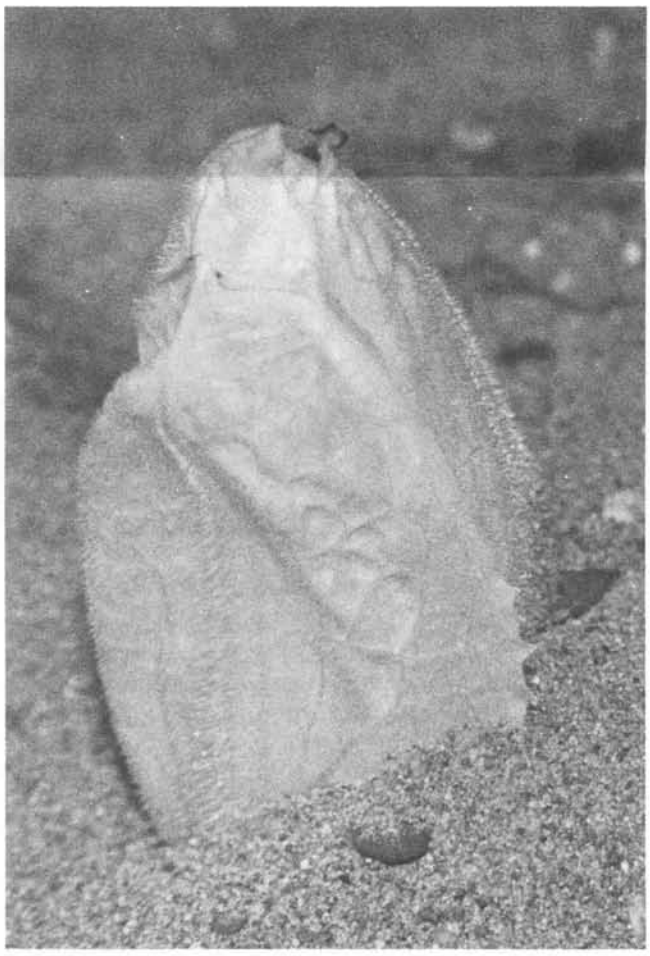

Fig. 1. Chicon emerging from a sand-peat mixture.

ground to dry for 2 days to lessen storage diseases. The wilted foliage was then severed, leaving a vegetative crown of 1 to 1½ inch long. The roots were trimmed to 8 inches, gathered, and stored in a darkened cold room at 35 F. For cultivars that can be forced early, temporary storage at 50 F is sufficient, but for roots held for mid- to late season forcing, cooling to 32-35 F is required. During cold storage, starch is reduced to simple sugars, which allows the roots to be forced.

The roots were forced in darkened, 3 x 12-foot chambers in a greenhouse or in a basement, which afforded more constant daily temperatures. Because several cultivars were grown, the roots could be forced in several ways.

Using the traditional European method, I planted some roots diagonally in soil to their crowns, watered them thoroughly and covered them with an additional 4 inches of soil. Twenty-five to 30 roots were packed into a square foot. The best roots for forcing were 1¼-1¾ inches crown diameter, although 1- and 2-inch roots were also used. Very large roots often produce multiple heads because they are usually over-mature. The loamy soil at Lockwood Farm produced about 15% more roots of optimal size than the sandy soil at the Valley Laboratory.

The soil for forcing was a 1:1 mixture of mason's sand and peat. New growth emerged from the crown and the tightly furled heads, called "chicons," pushed through the 4 inches of soil overburden. The crop was harvested when the tips of the chicons reached the surface. The chicons were severed from the roots and their outer leaves peeled to remove adhering sand and peat. Marketable chicons weighing an average of 80 grams (3 ounces) were produced in an average of 27 days with a soil temperature of 70 F.

Several cultivars have been developed in Europe to eliminate soil cover and reduce the cost of peeling and cleaning the chicons. These cultivars are planted diagonally in soil to their crowns and tightly furled chicons are produced without requiring soil cover. To employ this method, the air must be about 4 F cooler than the soil and the humidity must be at least 95%. I forced chicons in an average of 31 days by this method. The outer leaves were unfurled; removing them reduced the average weight to 60 grams.

Roots can also be forced hydroponically by placing them diagonally in about 3 inches of water fortified with a little calcium nitrate. I maintained the water temperature at 63 F with a heating cable, and a fish tank bubbler aerated the water. Under these conditions, forcing time increased to 43 days. Removal of unfurled outer leaves reduced the average weight to 50 grams. I also forced roots produced by a local grower. These roots, from another Dutch cultivar, produced between 71 and 96% marketable chicons. The roots of our Dutch cultivars produced between 45 and 70% marketable chicons. Some of the difference was due to the small, multiple heads on our over-mature roots.

For witloof chicory to be a profitable crop, the chicons must be of marketable size, furled, and withstand the rigors of display under normal lighting and handling by consumers. To see how Connecticut chicons would be accepted by consumers, I packed chicons in a standard 10-pound box and displayed them in a local supermarket. All chicons were purchased by consumers within 2 days and few outer leaves were bruised or rusted. Unfurling and removal of some outer leaves during the second day caused a loss of less than 5%. Some who tasted it found the Connecticut-grown chicory sweeter than the Belgian imports, which is undoubtedly due to the freshness of locally picked chicons that have not aged in transit from foreign lands.

EDB applied to soil not accumulated by plants

By Peter J. Isaacson and Charles R. Frink

Trace levels of a pesticide known as ethylene dibromide (EDB) have been found in ground water in Connecticut. Although EDB has not been used in agriculture since late 1983, concern arose over the possible contamination of vegetables planted in soil treated previously with EDB. Therefore, to simulate a worst case, we grew plants in soil soon after treatment with EDB, rather than waiting the normal four to six weeks for the EDB to dissipate.

We chose radish as the test plant because it grows quickly and has an enlarged root — the radish — which enhances the potential for uptake of EDB. Radish seeds were sown in plots at Lockwood Farm three days after application of EDB to the soil at the usual rate of 72 pounds per acre, which would produce a concentration of 35,000 parts per billion (ppb) in the soil. New seeds were sown at weekly intervals, and the plants were harvested 30 days after seeding.

The first crop of radishes grown in soil treated with EDB took up small amounts of pesticide. The concentrations at this first harvest, however, were less than the 30 ppb permitted by the U.S. Environmental Protection Agency for foods that are ready to eat. The average concentration of EDB in the radishes on a dry weight basis was similar to that in the soil, suggesting that EDB is not taken up preferentially by plants. Moreover, the concentration of EDB in the soil declined to only 0.4% of that applied at the beginning of the experiment. The concen-

tration of EDB in the radishes decreased steadily with time (Table 1) until none was detected in plants harvested 88 days after the soil was treated.

The radishes could either take up EDB in solution through the roots, or from the air through the leaves. Therefore, with the assistance of Martin Gent we conducted experiments in the laboratory to determine the amount of EDB which can enter the plant from solution and be transpired through pores (stomata) in the leaves. We also measured the amount of EDB that leaves may accumulate when exposed to its vapor in the air.

We measured transpiration by sealing radish leaves in a chamber with their stems protruding from the base into water containing 10,000 ppb of EDB. We sampled and analyzed air from the chamber at intervals over a three-hour period. We found EDB in the air in the chamber almost immediately, and the rate of transpiration of EDB increased to a maximum where the ratio of EDB to water in the air was close to that in the solution being supplied to the leaves. The actual concentration of EDB in the leaf, however, was less than one-tenth that in the water. This shows that EDB is readily lost from the leaves through the stomata.

We then measured the uptake of EDB from the air by exposing tobacco and radish leaves to EDB vapor in the chamber. Both tobacco and radish leaves took up EDB. The amount was regulated by the degree of opening of the stomata, which in turn was controlled by the amount of moisture in the leaf. For example, we found that wilted tobacco leaves with closed stomata took up one-third as much EDB as leaves adequately supplied with water. In the radishes, which closed their stomata only slightly when wilted, there was no decrease in uptake of EDB.

These experiments show that while plants can take up EDB from soil, water and air, it is not accumulated in the plant. Thus, it appears that plants grown in soil treated in previous years will contain little or no EDB.

Table 1. Concentration of EDB in radishes and soil

Days after EDB application to soil	Radish EDB (ppb)[a]		Soil EDB (ppb)[b]
	Leaves	Roots	
30	8.2	11.6	148
45	3.5	13.3	70
61	0.8	3.2	9.3
74	0.1	0.2	2.7
88	nd	nd	2.0

a) Fresh weight basis
b) Dry weight basis
nd = not detected.

No bee mites found in state-wide survey

By John F. Anderson

Already beleagured by bee diseases, wax moths and pesticides, Connecticut beekeepers have another threat on the horizon—*Acarapis woodi*, the honey bee tracheal mite (Fig. 1). This mite was discovered in England in 1921. Beekeepers and scientists believed then that it commonly devastated colonies of honey bees. Accordingly, the United States in 1922 enacted the Honey Bee Act to prevent the importation of live honey bees. Although this Act has been amended, honey bees from Europe and other continents still cannot be brought into this country unless there is substantial evidence that they are not infested with mites and are free of other diseases.

Studies in Europe in the 1950's and 1960's suggested that the honey bee tracheal mite may be less harmful than first thought. Although these investigations showed that infested bees died sooner than uninfested ones and that severely infested colonies (30% of the bees harboring mites) were more likely to die, most infested colonies were lightly infested and healthy. The mite is widely distributed throughout the world, but was first found in the United States last summer in several colonies in southern Texas.

Mites mainly infest the two large tracheae (air tubes) of adult honey bees. Female mites enter these air tubes only during the first nine days of the bee's adult life. Each female mite lays 5 to 7 eggs, which hatch in 4 to 6 days. Juvenile mites feed on body fluids and complete their development in 8 to 12 days. After mating, female mites crawl out of the air tubes to search for new hosts.

We examined honey bees in Connecticut last fall as part of a nation-wide survey to determine the location and

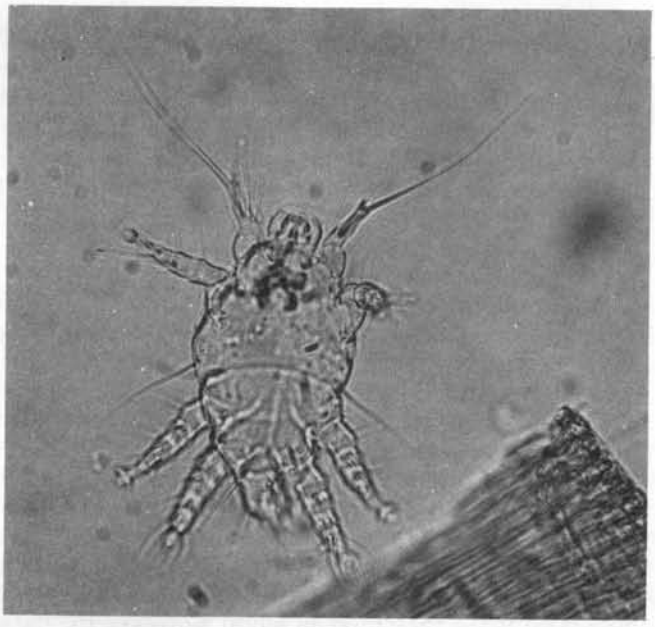

Fig. 1. Honey bee tracheal mite. This mite is from a honey bee collected in Texas.

prevalence of infested colonies. With the cooperation of beekeepers, honey bees were collected and stored in 70% ethanol from 52 apiaries in eight counties (Fig. 2). Both thoracic air tubes were dissected from each bee in the laboratory and examined for mites with the aid of a compound microscope. No mites were found in the 2481 bees examined. However, mites have been found in bees collected in ten states, including counties in northwestern New York State. This augers an uncertain future for the federal quarantine that began in 1922.

Although this mite will be difficult or impossible to eradicate from the United States, beekeepers can help prevent infestations by purchasing their bees only from sources that are certified to be free from this mite.

In response to a resolution passed by the Connecticut Beekeepers Association, the State Entomologist proposed and the Governor approved a state quarantine in April 1985 that prohibits entry of bees from states where infected bees have been found and requires that bees originating in uninfested states be certified free of tracheal mites before they are shipped to Connecticut.

Although the outlook for keeping this mite out of Connecticut is not promising, I close on this positive note: There is no published evidence that this mite has significantly harmed honey bee colonies in the United States.

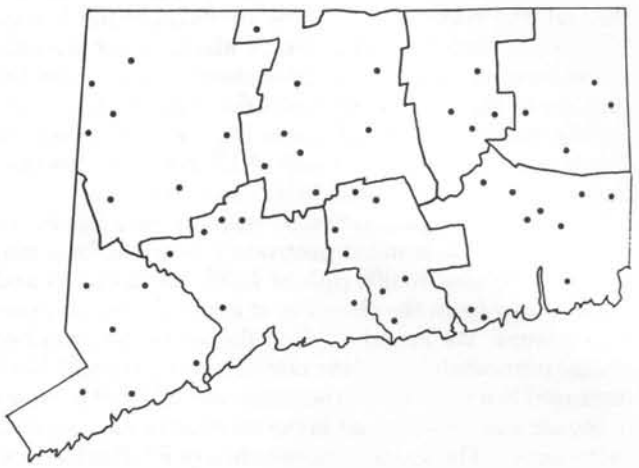

Fig. 2. Geographical distribution of apiaries sampled for honey bee tracheal mites.

Microsporidian parasites regulate mosquito populations

By Theodore G. Andreadis

Mosquitoes, like other organisms, are subject to fatal diseases caused by a variety of microorganisms, including bacteria, viruses, fungi, and protozoa. One of the most common microbial pathogens is a large group of obligate protozoan parasites called microsporidia. These one-celled pathogens are widely distributed and kill large numbers of mosquitoes. Although microsporidia have much potential to control mosquitoes, their development as microbial controls has been slow because their life cycles are poorly known.

I have been investigating these pathogens in mosquitoes for several years, concentrating on the genus *Amblyospora*. Members of this genus infect more than half the mosquito species in Connecticut. My studies show they are highly specialized parasites that take on different forms and have at least three separate and distinct developmental cycles, including two within mosquitoes and another within an alternate host.

Their life cycle is shown in Fig. 1. It begins in adult female mosquitoes, which harbor benign infections within specialized cells called oenocytes. Oenocytes are large cells found in fat tissue that are involved in molting. The females show no symptoms of disease and develop normally. When they feed on blood, however, the parasite transforms into a stage (binucleated spore) (Fig. 2A) that invades their ovaries and developing eggs. The next generation of mosquitoes is infected with the microsporidian through the eggs. This method of transmission of the parasite is called transovarial. In most species of mosquitoes the oenocytes of female larvae that hatch from infected eggs are again invaded by the microsporidian, which develops in a similar manner and has no adverse effect on the host. In male larvae, however, the microsporidian invades fat tissue, multiplies, and kills the mosquito. This developmental cycle produces many thick-walled spores (Fig. 2B) that, surprisingly, do not reinfect mosquitoes. The spores are haploid; they have only a single set of chromosomes. The function of these spores was, until recently, unknown.

In most mosquitoes transovarial transmission of microsporidia occurs with each host generation. This and the knowledge that haploid spores from male larvae were not infectious to mosquitoes, led investigators to believe

Table 1. Yearly rates of *Amblyospora* infections in larval populations of the salt-marsh mosquito, *Aedes cantator*.

Year	% infection in each brood		
	spring	summer	fall
1982	1.5	1.0	98.1
1983	1.1	4.0	14.3
1984	1.3	2.2	97.4

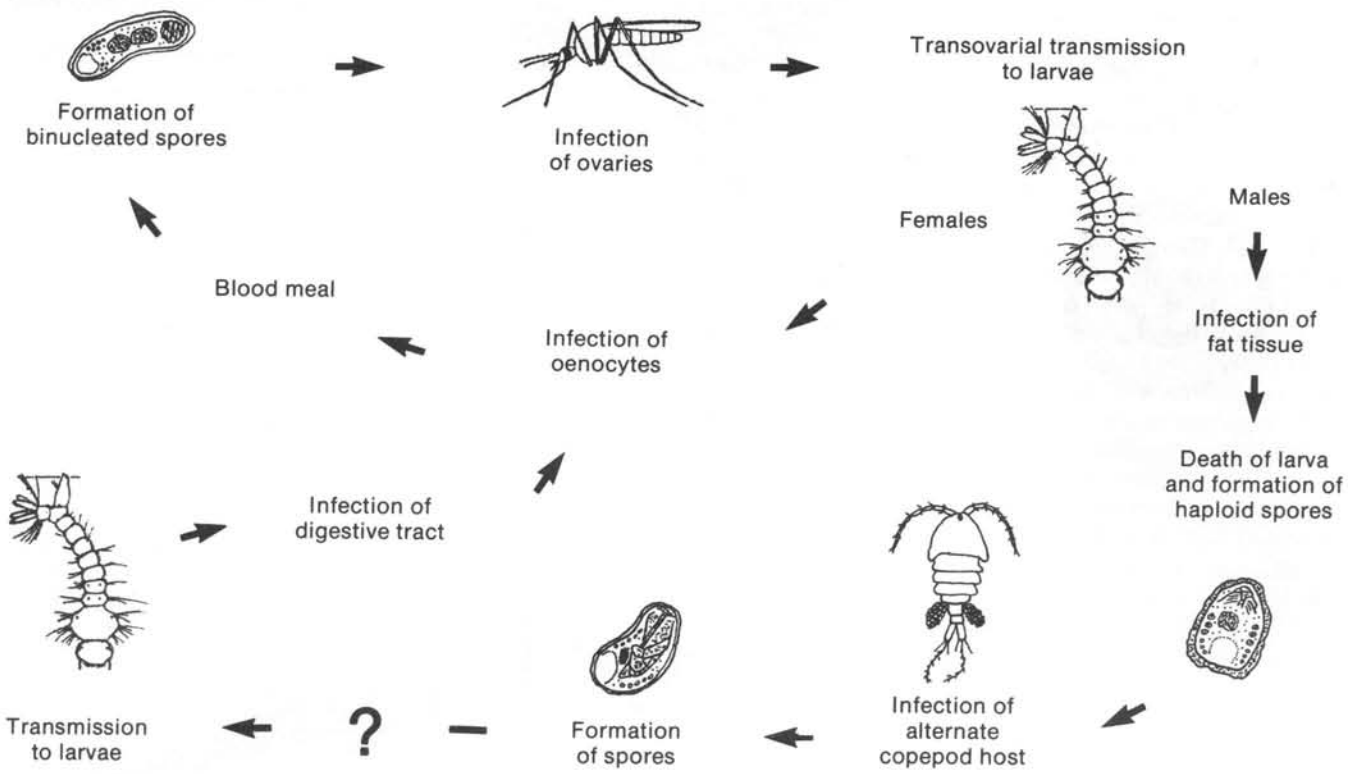

Fig. 1. *Amblyospora* life cycle.

that transovarial transmission was the only way these pathogens spread among mosquitoes.

Recently, however, I discovered in many mosquitoes that infections are transmitted to only half the female progeny. In males, the effects of the parasite can be seen in the larvae. In females, however, I must wait until adults have mated, and then catch females and bring them back to the laboratory where they lay eggs in separate cages. Dissection of the female shows if she was infected. I save the eggs in a refrigerator for several months to simulate the cold weather that is necessary to stimulate the eggs to hatch. Studies of the larvae that hatched from these eggs showed that transovarial transmission did not occur at a rate that could maintain the

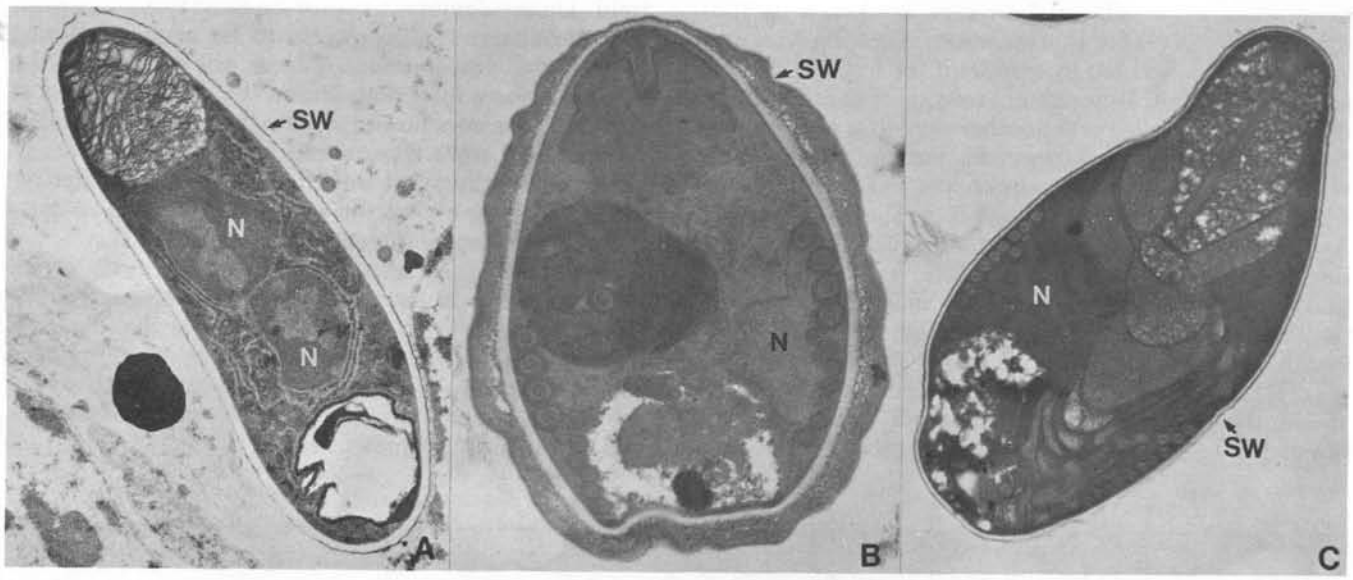

Fig. 2. Electron micrographs of *Amblyospora*. (A) Binucleated spore from an adult female mosquito X 13,500. (B) Thick-walled haploid spore from a male larval mosquito X 15,300 (C) Uninucleated spore from a copepod X 9,500. (N, nucleus; SW, spore wall).

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pathogen in natural mosquito populations. Therefore, I concluded, there must be another method of transmission.

Continuously monitoring microsporidian infections in the salt marsh mosquito *Aedes cantator*, I discovered that the level of disease increases dramatically in the fall generation; up to 98% of the larvae are killed by the microsporidian (Table 1). This discovery, along with my previous work, led me to wonder if the haploid spores from male larvae, although not infectious to other mosquitoes, might develop in another organism into a form that could later infect mosquitoes. Such a phenomenon was reported in another mosquito species in Australia, so I began to investigate this possibility. The other host in Australia was a copepod, a small crustacean that is related to shrimp (Fig. 1). Therefore, I collected copepods from the pools where salt marsh mosquitoes develop and demonstrated in the laboratory that haploid spores from the male larvae would infect copepods (Fig. 1). Because the phenomenon occurs in two places among different species of mosquitoes and copepods, this method of infection may occur in still other mosquitoes. I have also found that the microsporidium underwent another cycle of development in the copepod that formed a third type of spore (Fig. 2C).

At the same time, I also observed a distinct and previously unknown type of benign infection within the digestive tracts of larval mosquitoes breeding in the field. These infections were unlike those from transovarial transmission and appeared to be acquired during early larval development. I have not yet proven the source of these infections within the digestive tracts of larvae. These infections spread to the oenocytes in adult females and were transmitted transovarially (Fig. 1). Thus, this pathway of infection through the digestive tracts of larvae allows the microsporidium to re-enter the mosquito population and perpetuate itself.

To complete the unraveling of the life cycle of the microsporidian in mosquitoes, I have only to find the agent that causes the infections within the digestive tract, which I suspect is the spore from the copepod. I can now begin to direct my attention to finding ways to use these pathogens to control mosquitoes in salt marshes and woodlands in Connecticut.

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