

Diseases and Pests of Honey Bees

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BEEKEEPING IN THE UNITED STATES
AGRICULTURE HANDBOOK NUMBER 335
Revised October 1980
Pages 118 – 128

The first bee laws in the United States were enacted in 1883 to establish methods for control of bee diseases. Today, 49 States provide apiary inspection services for disease abatement. Bee diseases cause considerable expense to the States for the cost of maintaining apiary inspection service, as well as considerable losses to the beekeepers for the cost of colonies damaged or destroyed and for the drugs fed to prevent bee diseases. In addition, far greater losses result from reduction in honey and beeswax production and insufficient bees for pollination. It is apparent, therefore, that both beginning and advanced beekeepers should learn to recognize and control bee diseases.

Brood Diseases

The most common brood diseases found in the United States are American and European foulbrood, sacbrood, and chalk brood. A guide for diagnosing brood diseases of honey bees is given below.

American Foulbrood

American foulbrood disease occurs throughout the world where honey bees are kept. About 3 percent of all colonies inspected in the United States are found to be infected.

Bacillus larvae White, the causative organism of American foulbrood disease, is a spore-forming bacterium which produces over a billion spores in each infected larva. Only spores are capable of inciting the disease. The spores are extremely resistant to heat and chemical agents. Worker, drone, and queen larvae are susceptible to the disease. Under natural conditions, infected queen and drone larvae are rarely seen.



A severely infected American foulbrood comb has a mottled appearance due to a mixture of healthy capped brood, cells containing the remains of diseased larvae, and empty cells. The cappings of cells containing disease appear moist and darkened. The convex cappings found on cells of diseased larvae become concave as the disease progresses. Another symptom commonly associated with the disease is the punctured capping. Larvae are susceptible to American foulbrood only when they are less than 3 days old. A healthy larva has a glistening, pearly white appearance. Normally it begins development curled on the base of the cell. As it grows, it elongates to the full length of the cell. It is in the elongated position that the larva or pupa dies. As the infection progresses, the larva or pupa changes to creamy brown and eventually becomes dark brown. The remains become ropy and can be drawn out as threads of an inch or more. A very unpleasant, foul odor develops at this stage. The odor resembles that of animal glues that are rarely used.

The remains of diseased brood finally dry down to form scales that adhere strongly to the lower sides of the cells. If death occurs in the pupal stage, the mouth parts may adhere as a fine thread to the upper side of the cell. This is a positive symptom of American foulbrood disease.

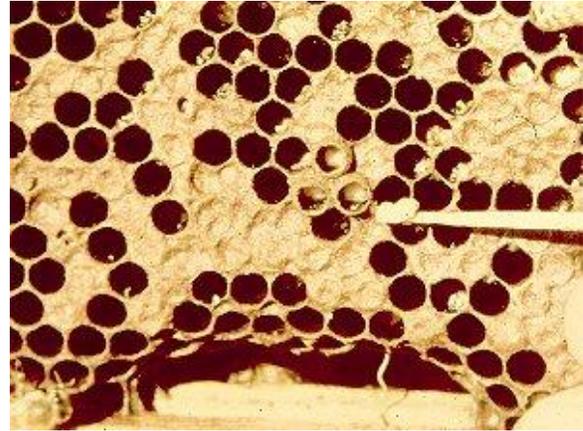
The infection can be transmitted to a larva from nurse bees or from spores remaining in the bottom of the brood cell. Exchanging combs containing remains of diseased larvae or honey, or both, laden with spores of *B. larvae* is the most effective way to spread the disease from colony to colony. Early detection of the disease is helpful in preventing further spread. A colony that is weakened by American foulbrood may be robbed, and the robber bees inadvertently carry honey containing spores of *B. larvae* to healthy colonies.

European Foulbrood

In some areas, European foulbrood is a more serious threat to beekeepers than American foulbrood. This disease is serious because it occurs most frequently at the time that colonies are building their peak populations.

The cause of this disease is *Streptococcus pluton* White, a nonspore-forming bacterium. Other bacteria commonly associated with the disease are *Bacillus alvei* Chesire & Cheyne and *Bacterium eurydice* White.

Superficial examination of diseased combs shows the same mottled effect and puncturing as seen in American foulbrood. Death usually occurs in the larval stage. Worker, drone, and queen larvae are equally susceptible to European foulbrood.



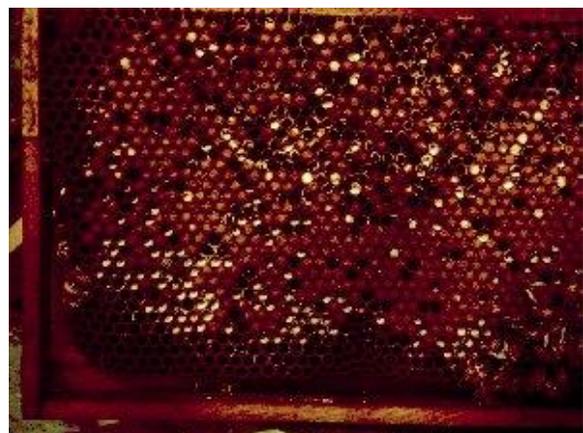
Larvae that die from European foulbrood are found in various positions. Some are in a curled stage and others elongated. The normal pearly white appearance of a healthy larva changes to a dull white, then yellow and finally brown. Ropiness and sour odor are caused by the secondary organisms associated with the disease. The elasticity of the ropy material is less than that associated with American foulbrood. The tracheae appear as fine silvery tubes immediately below the skin, especially as the larvae turn brown. This symptom is highly characteristic of European foulbrood. Loosely adhering scales also differentiate this disease from American foulbrood.

European foulbrood can be transmitted by contaminated food stores, and equipment. The disease usually is most serious in the spring and clears up during the summer when nectar and pollen are abundant. However, outbreaks of European foulbrood in the late summer are not unusual.

Chalk Brood

Chalk brood disease was not found in this country until 1968. Since that time, the disease has spread throughout the United States and Canada. No accurate figures are available on losses attributed to this disease. Chalk brood appears primarily in the spring, although outbreaks in the summer and fall can occur.

Ascosphaera apis (Massen ex Claussen) Olive and Spiltoir, a fungus, is the cause of chalk brood disease. Larvae, 3 to 4 days old, are most susceptible to the



fungus, especially if they are chilled after ingesting spores of *A. apis*. Worker, drone, and queen larvae all are susceptible to chalk brood disease.

Infected larvae become permeated with the mycelia of the fungus, leading to their death. Eventually, the mycelia-filled larvae dry up to form the typical hard white mummies characteristic of chalk brood disease. Diseased larvae are stretched out in their cells and also can be mottled or completely gray or black. This color variation is due to the presence or absence of the black fruiting bodies that are formed on the outside of the larvae.

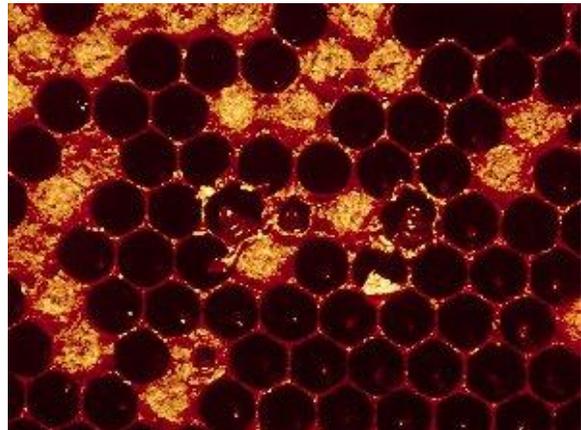
The disease can be detected by examining the combs, the entrance, and bottom boards of the hives for the presence of the mummies. The mummies do not stick to the cells and are easily removed by nurse bees. If colonies have pollen traps, the mummies frequently are found in the traps and are a source of infection in trapped pollen. The mummies have a faint yeast-like odor.

Chalk brood disease can be transmitted by adult bees and equipment contaminated with spores of *A. apis*. The disease appears to clear up spontaneously and may reappear later in the season or the following year. The disease rarely destroys a colony but can reduce the population of bees and consequently affect the honey yield.

Sacbrood

Death of a colony by sacbrood is rare. Because of the similarity to other diseases, however, the beekeeper should learn to distinguish sacbrood from the more serious diseases. The etiologic agent in sacbrood is a virus.

Larvae die of sacbrood in capped cells in the elongated position. As the disease progresses, the larval skin forms a sac, which separates from the prepupal skin. Between these two layers of skin is an accumulation of fluid. The outer skin toughens and, as a result, the larva can be picked up in its entirety without the release of the fluid.



The larva changes from pearly white to offwhite, then brown, and finally almost black. The head of the larva usually curls up from the cell floor. A loosely adhering scale is formed from the larval remains. It has the appearance of a foulbrood scale but no odor, and is free of bacteria.

Like European foulbrood, sacbrood is most commonly found in the spring. No chemotherapeutic agent is effective against sacbrood. Requeening may be helpful, but most colonies appear to recover spontaneously from the disease.

Other Blood Diseases

Aspergillus flavus Link, a fungus, usually is isolated from bees that have stonebrood. This disease is unusual in that it infects both brood and adults. Bees dying from this disease form mummies. The fruiting bodies of the fungus make the infected bee appear yellowish-green or brown.

Purple brood is a nutritional disease of larvae and pupae. It is believed to be caused by nectar or pollen from *Cyrilla racemiflora* L., also called southern leatherwood, black titi, red titi, summer titi, and he-huckleberry. This problem exists only in the Southern States, where southern leatherwood is found. Diseased larvae and pupae are purple.

Other conditions that mimic contagious diseases are chilled and starved brood. Chilled brood is caused by lack of sufficient bees to keep the brood area warm. Consequently, chilled brood usually is found at the outer edge of the brood nest. Brood may have the appearance of European foulbrood but is readily removed by nurse bees as the brood pattern expands. Starved brood generally is caused by insufficient nectar or honey. At times, wax moth damage to developing bees may cause them to appear diseased.

Adult Diseases

Nosema Disease

Nosema disease is the most widespread of all bee diseases. It was found in over 60 percent of the apiaries sampled in the United States. This disease is caused by the protozoan, *Nosema apis* Zander. Nosema disease reduces the life expectancy of adult bees. It can cause queen supersedure and reduce the honey production of infected colonies.

The disease cycle is initiated by adult bees that ingest spores of *N. apis*, which germinate and multiply in the epithelial cells of the ventriculus. In addition to affecting the digestive process, the hypopharyngeal glands of infected worker bees and the ovaries of infected queens become atrophied. The disease is found in workers, queens, and drones.

No external symptoms may be visible in bees or in colonies infected with *N. apis*. Some of the infected ventriculi may become distended and white. Nosema disease is diagnosed by examining for the presence of *N. apis* spores. However, the absence of spores does not ensure freedom from nosema disease, since other life stages of the protozoan may be present. The disease has an annual cycle which results in maximum numbers of spores in the spring. Spore numbers decline in the summer, and in some cases, a small peak in the fall may be visible.

Virus Bee Paralysis

Bee paralysis is caused by several different viruses, but some nectars and pollens also may induce similar symptoms. Chronic bee paralysis and hairless black syndrome are caused by the same virus. Acute bee paralysis, caused by another virus, kills bees more quickly than the chronic virus.

Affected bees quiver and cannot fly. Frequently, they appear greasy and shiny with no hair on their thorax. The disease is transmitted to healthy bees when they attack diseased bees or when food is exchanged between healthy and diseased bees.

Workers, drones, and queens are susceptible to chronic bee paralysis. It appears that susceptibility to the disease is inherited from the queen. Consequently, requeening of colonies with the disease may rid the colony of all symptoms.

Septicemia

Septicemia is a bacterial disease of adult honey bees that is rarely encountered; it is caused by *Pseudomonas apisepctica* Burnside.

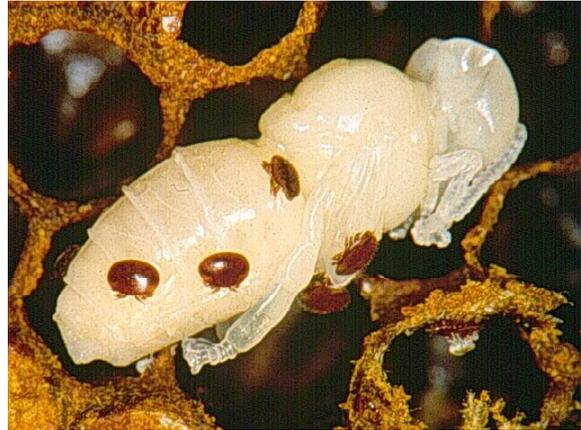
The bacteria, by some unknown method, make their way to the hemolymph, multiply rapidly, and ultimately cause the death of the host.

Bees that die from septicemia appear to have no connective tissues and dismember easily. The legs, wings, head, thorax, and abdomen separate-even by the slightest handling. Hemolymph of infected bees also may be milky white in color. The isolation and identification of *Ps. apisepctica* bacteria from the hemolymph may be necessary for verification.

Mite Diseases

The Asiatic mites, *Varroa jacobsoni* Oudemans and *Tropilaelaps clareae* Baker and Delfinado, both affect larvae and pupae of the honey bees. *Varroa jacobsoni* has been found in South America but is not present in North America. However, *T. clareae* has not been found on bees outside Southeast Asia.

The mature female *V. jacobsoni* mite attaches itself to bees and can be transmitted from colony to colony by robbing and drifting bees. The female mite lays her eggs in cells containing larvae just before being sealed. After the eggs hatch, the nymphs feed on the developing larvae or pupae, causing malformation and sometimes death of the host.



Acarine Disease

Acarapis woodi Rennie, the causative agent of acarine disease, has not been found in North America. The Honeybee Act enacted in August 1922 and amended several times since prohibits the importation of all live stages of the honey bees and was written principally to prevent the entry of *A. woodi* into North America.

The mite enters its host via the spiracles and spends most of its life in the thoracic tracheae. At maturity, the female mite emerges from the tracheae in search of a new host. No one symptom characterizes this disease; an affected bee could have disjointed wings and be unable to fly or have a distended abdomen, or both. Positive diagnosis of this disease can be made by microscopic examination of the tracheae for discoloration (black spots) and the presence of eggs, nymphs, and adult stages of the mite.

External Mites

External mites frequently are present on bees in the United States. These mites closely resemble *Acarapis woodi* but cause no apparent harm to the bees. A microscope is necessary to differentiate external mites from *A. woodi*.

Other Adult Diseases

Amoeba disease and gregarines are protozoans sometimes found in honey bees in North America. Neither disease seems to be of economic importance.

Amoeba disease is caused by *Malpighamoeba (Vahlkampfia) mellificae* Prell. This disease sometimes is found together with nosema disease, and the combination may be more serious than either disease alone. The cysts of the amoeba are transmitted by the excreta of bees, and the infection is localized in the Malpighian tubules. It is believed that the amoeba interferes with the function of the Malpighian tubules, which ultimately leads to the death of the bee. Diagnosis for this disease is made by microscopic examination of the Malpighian tubules for the presence of amoeba cysts.

Gregarines are found in the digestive tract of adult bees. No pathological significance, however, has been attached to these protozoans. Several different genera of gregarines are found in the United States.

Wax Moths

The most serious pest to honey bee colonies is the greater wax moth, *Galleria mellonella* L. In addition to the greater wax moth, comb damage is caused by the lesser wax moth, *Achroia grisella* F. and the Mediterranean flour moth, *Anagasta kuehniella* Zeller.

Damage by the greater wax moth is severest in the Southern United States because of the long warm season and high temperatures. The wax moth distribution, however, includes all areas where honey bees are kept. It is not a threat to normal colonies and cannot kill a colony, but weakened colonies are invaded and unused combs destroyed.



Female wax moths lay their eggs on combs or in cracks between the wooden parts of the hives. After egg hatch, the larvae feed on the wax combs, obtaining nourishment from the cast-off honey bee pupal skins, pollen, and other impurities found in the combs. For this reason, darkened combs are more likely to be infested than light combs or foundation.

The fully grown larva spins its own cocoon, which usually is attached to wooden parts inside the hive—such as the inner cover, hive body, and frame. In colder climates, the greater wax moth overwinters as a pupa. In warmer areas, adults emerge all year. The adult female is about 3/4-inch long and 1 to 1-1/4 inches wide from wingtip to wingtip. Within 4 to 10 days after emergence, the female begins to lay eggs. She lays about 300 eggs in her lifetime, which usually is somewhat less than 3 weeks.

Combs are most often destroyed by the wax moth when stored in dark, warm, and poorly ventilated rooms. However, there can be considerable damage to combs even while in use, especially in hives where the population of adult bees is too small to protect all the combs.

Paradichlorobenzene and ethylene dibromide have been used in the past for the control of wax moths. Other control measures include carbon dioxide, heat, or cold treatments.

Larvae of the Mediterranean flour moth and the lesser wax moth also can cause damage to combs in storage and in the hives. The damage caused by these insects is quite similar, and it is necessary to identify the insects to be certain which is causing the problem. The same control methods work for all three insects.

Other Insect Pests

In some areas of the United States, termites may damage the wooden parts of the hive. The termites do not affect the bees directly. Although ants can be found in hives, they rarely cause any problem. Ants are more of a nuisance to the beekeepers than to the bees. Other insects such as certain wasps and robber flies also prey on honey bees but are considered of no economic importance in the United States.

Braula coeca Nitzsch, the “bee louse,” is found primarily in the Mid-Atlantic States. The bee louse actually is a wingless fly and not a louse. This insect can be found on drones, workers, and queens. The destructive stage of the insect is the larva, which burrows under the cappings of honeycombs and ruins what would be good comb honey.



No apparent damage is attributed to the adult bee louse, which spends its life on the bodies of workers and queens.

Enemies of Bees

Bears cause severe damage to the hives as they feed on the honey, adult bees, and brood. Electric fences and bear platforms have been used successfully to prevent bear damage.

Skunks, birds, toads, and frogs also feed on adult bees. Skunks can cause serious damage, as they consume large numbers of bees and thereby deplete populations to critical levels.

In the winter, mice can enter hives without entrance reducers. Although mice do not feed on bees, they chew on the combs and frames to construct nests in a warm, secluded area of the hive where they are not disturbed by the clustered bees.



Control of Bee Diseases

The U.S. Food and Drug Administration approved labeling for oxytetracycline and fumagillin as aids in the control and prevention of bee diseases. Oxytetracycline is effective in the control of both American and European foul brood disease. Fumagillin is used to control nosema disease. Users should read the container label-it has specific instructions for the use of these materials. These drugs can be used subject to State laws and regulations in the manner specified but should never be used at a time or in such a way that would result in contamination of the marketable honey. Some States require that the bees, contaminated combs, and honey from infected colonies be destroyed by burning. Beekeepers should consult their local apiary inspector for instructions on the disposal of diseased hives and the use of drugs.

Sending Samples for Laboratory Examination

If only a small amount of the brood or a few bees are affected or if the symptoms are unusual, a definite diagnosis in the apiary is sometimes difficult. Examination by laboratory methods is then necessary. Sometimes laboratory verifications of diagnoses made in the apiary also are desirable.

Diagnosis of disease in the laboratory is a service made available to beekeepers and State apiary inspectors by the U.S. Department of Agriculture.

A sample of brood comb for laboratory examination should be 4 or 5 inches square and contain as much of the dead brood as possible. *No honey should be present*, and the comb should not be crushed. A sample of adult bees should consist of at least 200 sick or recently dead bees.

Mail the samples in a wooden or strong cardboard box. Do not use a tin, glass, or plastic container, and do not wrap the comb or bees in waxed paper or aluminum foil. Send all samples to the U.S. Department of Agriculture, Science and Education Administration, Bioenvironmental Bee Laboratory, Building 476, BARC-E, Beltsville, Md. 20705. Your name and address should be plainly written on the box. If the sample is forwarded by an inspector, his or her name and address also should appear on the box.