

A General overview on AFB and EFB Pathogen, Way of infection, Multiplication, Clinical symptoms and Outbreak

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About two thousand years ago, in times of Varro, Virgil, Columella, the first descriptions of several bee diseases were published, two of them probably being the American and European foulbrood (CRANE, 1994). We assume thousands of years of co-evolution between the bacterial pathogens and the honey bee *Apis mellifera*.

Today much more is known about these brood diseases, leading to more questions. The two key questions are concerned with the causation of the outbreaks and the preventions of such without using chemical interventions.

European foulbrood

Pathogen and clinical symptoms

European foulbrood (EFB) is caused by the bacterium *Melissococcus plutonius*. The symptoms vary, as other types of bacteria are often present in the effected brood. Larvae die at the age of four to five days, rarely in capped cells.

Larvae could be twisted and their colour will change from pearly white to yellow and brown, finally greyish black (RITTER, 1996).

EFB has been known for a long time in Europe and North America. More recently it was also found in Africa, South America, India, Japan and Australia (MATHESON, 1995). EFB appears less frequently than AFB and does not cause such damages as American foulbrood does.

American foulbrood

Pathogen and clinical symptoms

American foulbrood is the most widespread and damaging of the brood diseases. Only the brood is affected, whereas adult bees are insensible.

The disease is caused by the spore-forming bacterium *Paenibacillus larvae* larvae WHITE, formerly *Bacillus larvae*. It occurs in two forms: vegetative (rod-shaped bacterial cells) and spores. Only the spore stage is infectious to honey bees. The spores are very resistant to heat and to most of the chemical disinfectants. They can probably survive hundreds of years.

Paenibacillus larvae larvae characteristically produce microscopically visible flagella accretions. (GOCHNAUER and L'ARRIVE, 1969) (fig. 1). Additional diagnostic methods of cultural, morphological, biochemical as well as DNA testing are described to detect *Paenibacillus larvae* larvae.

Larvae affected with *Paenibacillus larvae* larvae usually die in an upright position when the cells are sealed. Cell cappings become punctured, sunken and moist, because house bees try to clean them (fig. 2). On insertion of a matchstick one can find a slimy, brownish viscous mass (fig. 3).

However, dead larvae dry out and become a brittle scale. It is easier to recognise them on new, light combs, rather than on old dark combs (fig. 4). The flat scale typically extends from the inner comb foundation to the front edge of the brood cell and adheres tightly to the cell wall. STURTEVANT (1932) stated that one scale could contain more than two billion spores.

Infection

The infection of colonies continues to be a problem and is most often caused by :

- using contaminated equipment
- change of infected brood combs between colonies
- feeding contaminated honey
- inserting honey combs that had contained contaminated honey
- robbing bees, which transfer great amount of spores from diseased, weak colonies to their own. (VON DER OHE and DUSTMANN, 1996; HANSEN and BRØDSGAARD, 1999)

On transferring the spores - for example by robbing bees - they will be transmitted by trophallaxis from the robbing foragers to house bees and nurse bees. Last will feed the contaminated honey to the brood.

Today we know that only the youngest larvae, one to two days old, react susceptible. After oral infection, spores germinate in the midgut of the youngest larvae, multiply and penetrate to the body cavity through the midgut wall. About six to seven days later, after the cells are capped the larvae die and new spores are formed. The mass of the former larvae dry out and the described scale develops. On cleaning these cells, the house bees distribute the spores inside the whole colony. The vicious circle is now closed. If the mechanisms of resistance of the sick colony are too weak more and more larvae become infected and the colony will finally break down. It is now, that strange bees from other colonies can invade and contaminate themselves.

Distribution

With some exceptions and in contradiction to its name, the American Foulbrood is found world-wide and an increasing number of countries are affected. However, not in all of the affected countries colonies spores or colonies with clinical symptoms could be proven.

In Germany, there are about one million bee colonies in approximately hundred thousand apiaries. Of these apiaries about 250 to 350 are effected by AFB each year (OTTEN, 1991; 1993). Noticeable are the regular, periodical ups and downs of outbreaks in Germany in the last 50 years (fig. 5). Until now, no background could be given for this phenomenon.

However, not all colonies in the effected bee yards show the disease or were infected.

Investigations in the German states of Rheinland-Pfalz and parts of Nordrhein-Westfalen showed that in random samples only 6 percent had spores of *Paenibacillus* larvae larvae, 4% of a low level and 2% on a high level. In suspicious areas, the amount of contaminated samples was about 24 %, 18% on the lower level and 6% on the higher level. Fig. 6 shows numerous 'hot spots' that were found around AFB bee yards

Similar results for other regions of Germany have been reported by RITTER (1993) or VON DER OHE et al. (1996).

Outbreak

There are three main factors causing an outbreak: the severity of infection, the resistance of *Apis mellifera* and probably the pathogeneity itself (fig. 7).

In 1942, WOODROW stated that only a few spores were necessary to infect one young larvae within the first 24 hours. Several other authors described that an outbreak is caused by about 50 Million and two billion spores per colony (HANSEN and BRØDSGAARD, 1999)

The range within the susceptibility could be explained by different resistance of the investigated colonies. The resistance itself is influenced by genetic and environmental conditions, for example the strength of the colony, the hygienic behaviour, physiological characteristics, antagonism mechanisms of micro flora (REICHE et al., 1996) or nectar flow or the possibility to collect pollen.

On the other hand we know that there are different strains of *Paenibacillus larvae* larvae (JELINSKI, 1985; REICHE et al. 1997; OTTEN et al. 1998 and we assume that there are differences in virulence.

Prevention

From the practical point of view, it is important to interrupt the transfer of spores between colonies. This seems to be difficult. Experience has shown that only about 40% of all beekeepers are able to recognise the symptoms of AFB or they recognise them too late (OTTEN and RITTER, 1995). Meanwhile great amounts of spores could be transferred to other colonies or apiaries by robbing bees. To put a stop to this way of infection the awareness of beekeepers has to be improved.

The second and probably better way of prevention is the early detection by honey and wax analysis to initiate control measures (HANSEN, 1984; RITTER, 1990, 1993; VON DER OHE, 1996).

Conclusion

To get healthy colonies without the treatments of chemicals such as antibiotics or other drugs we should

- support the resistance of *Apis mellifera*
- establish methods for early detection of AFB and EFB
- improve the awareness of beekeepers

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Fig.1



fig.2



fig.3



fig.4



fig.5

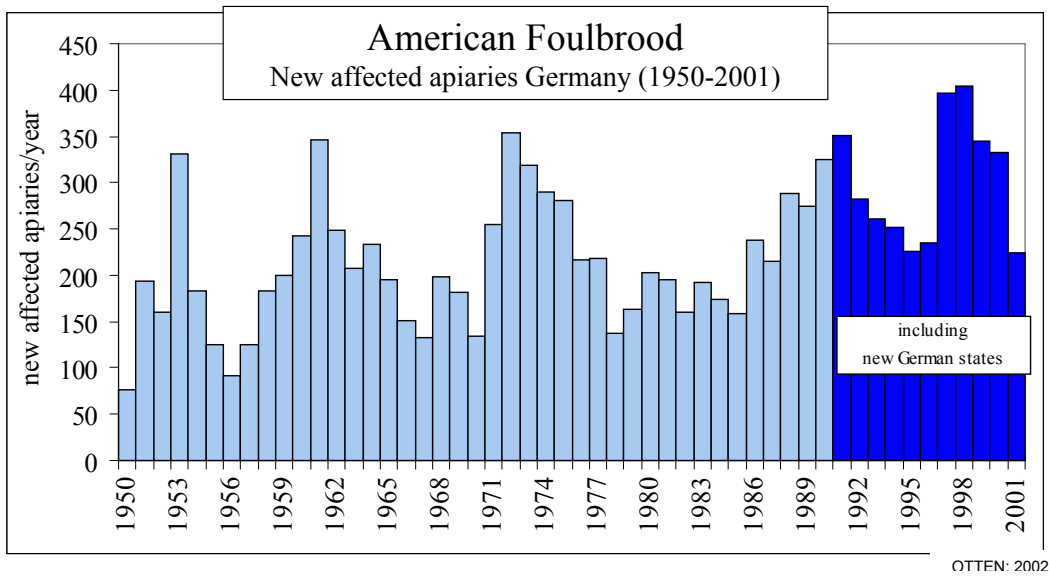


fig.6

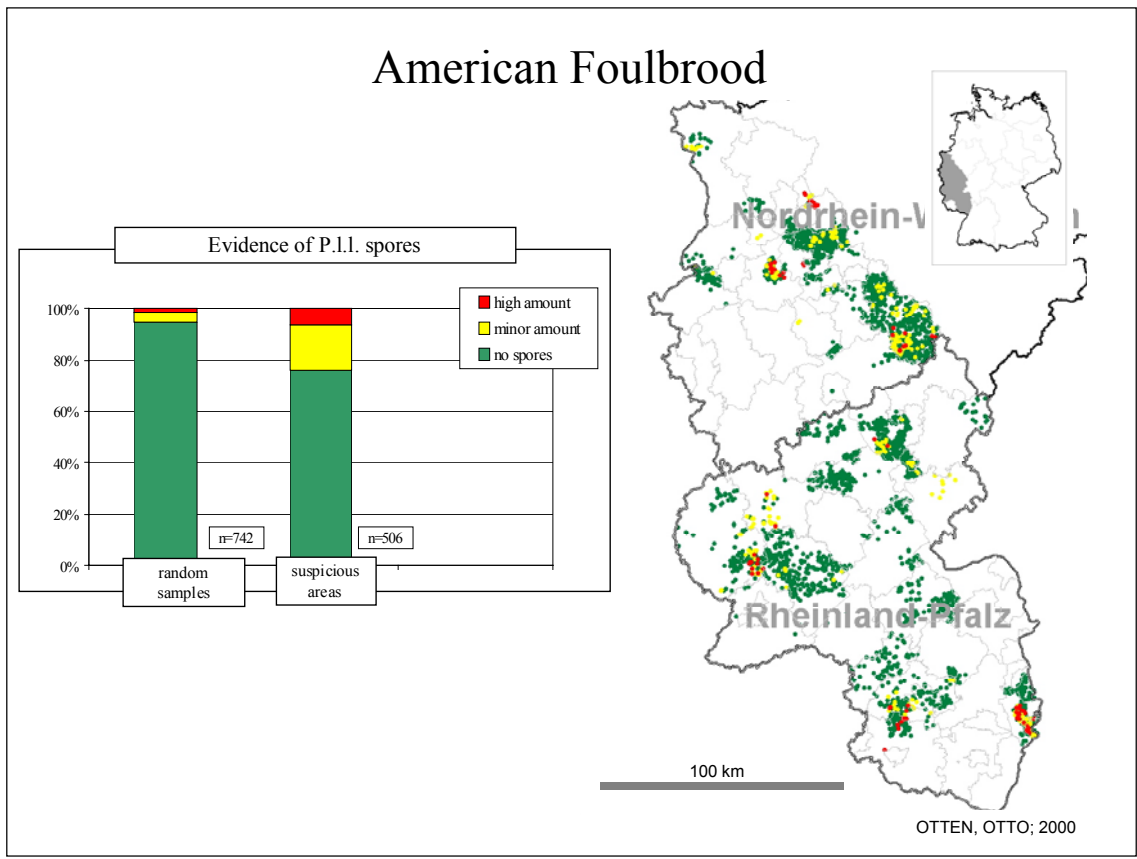


fig.7

