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A BIBLIOGRAPHIC REVIEW ON THE MOST DANGEROUS DISEASES OF THE HONEY BEE

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Abstract

Honey Bees, in addition to their production of honey, pollinate fruit trees and other crops to flowers. Any threat to bees, whether from herbicides, pesticides, diseases or parasites is fraught with consequences not only for the bee, but also for agriculture in general.Numerous reports and studies have reported recent deaths and massive loss of bee colonies (Colony Collapse Discorder). The health of bees is one of the factors that causes this phenomena. The purpose of this article is to review a recent literature on major diseases of bees in different stages of their development.The study presents a synthesis on the main diseases: varroasis and acariosis, American and European foulbrood and viruses. All these pathologies have a negative influence on the colonies, causing symptoms and a decrease in production and mortality

Keywords: Honey Bees; herbicides; pesticides; mortality.

1. INTRODUCTION

The bee'sessential factor in the environmentalbalance inthe world for its role in the pollination of many plantspecies.**It also has other interests such as the production of honey, propolis, royal jelly and wax**. It is noteworthy that in recent years, impairments of hives are identified in many countries. This phenomenon results in a winter mortality of bee colonies than normal and population losses during the year. This weakening is known as the '' Colony Collapse Disorder '' (CCD). These losses are estimated at 40% on average in Austria, Belgium and Switzerland(Haubruge et al., 2006). It has beenthe same in France every year since 1995 a loss in colonies of between 300 000 and 400 000 is registered (Guillet, 2007). In Canada, a 21.3% reduction is observed in winter 2009 (Boucher, 2009). During the period 2009-2010, 33.8% of the American colonies have disappeared (Van Engelsdorp et al., 2010). In Japan, the

number of colonies was reduced by 25% (Neumann 2010).The decline in honey production varied between 20 and 30% between 1997 and 2009 because of this phenomenonof Colony Collapse Disorder (Genersch et al., 2010) . Several studies have beenconducted on the causes of these impairments and involve a variety of pathogens (Boucher and Desjardins, 2005; Ellis Et Munn, 2005; Oldroyd, 2007; Burgett Et Al., 2009; Currie Et Al, 2010.;Novoa Guzmanet al, 2010;. NeumannandCarreck, 2010; Adjlane et al., 2012a; Jabaji Copley, 2012).

This article aims to make a general summary of the most dangerous bee diseases affecting the honey bee.

2. ACARIOSIS

In 1921, in England, this disease was identified for the first time. Acariosis is a contagious parasitic disease of the respiratory system of adult bees. It is caused by a microscopic mite *Acarapiswoodi* (Rennie, 1921). *Acarapiswoodi* infests bee three castes: workers, drones and queens (Bailey, 1985). The mite is an internal parasite that lives, feeds and breeds in the respiratory system of the bees. All stages of development and will live primarily in the first pair of chest tracheas (Delfinado-Baker and Baker, 1984) (Figure 1). The life cycle of this mite takes place entirely in the tracheal respiratory system of adult bees except for short migratory periods. Within 24 hours of discharge from the bee from its socket, mites adult females will enter the trachea by passing through the thoracic spiracles and will remain there until the death of their host. The females of the mite, after being fertilized inside the trachea will seek another bee on which they will migrate (Giordani, 1965). The pathogenic effects found in infected bees depend on the number of parasites in the trachea and are attributable to the mechanical damage and the physiological disorders consecutive to the obstruction of the air ducts, the lesions in the walls of the trachea and the reduction of hemolymph (Otis and-Scott Dupree, 1992). BAILEY in 1961 showed that the parasitism reduces the life of the bees. Those that are parasitized will die before those who are not, or will result in a significant loss of production. The predatory mite action paves the way for secondary infections (Eischen et al, 1989) and also causes a loss of nutrients for the bees (Eischen 1987). A careful examination of the trachea is required to determine the mite infestation. There is no effective treatment to 100% for acariose. Once this disease in the apiary, the beekeeper must live with and control its development to a level that will not affect the health of the colony. Several products can treat this parasitic infestation: menthol, thymol, formic acid and chemicals such as amitraz, fluvalinate and flumethrin (Dawicke et al., 1992).

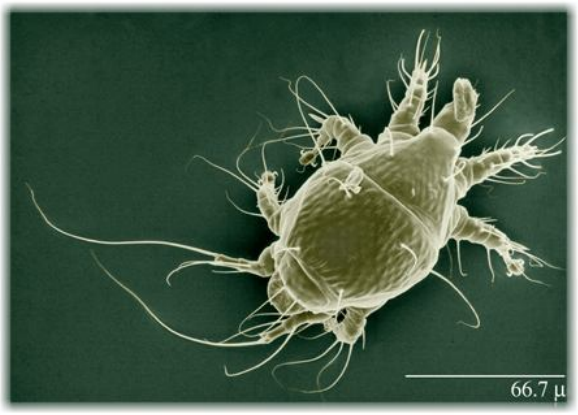

Figure 1: *Acarapis woodi* observed with an electron microscope (Delfinado-Baker and Baker,1982)

3. NOSEMOSIS

Nosemosis is a disease caused by microsporidia *Nosema* the kind that affects the digestive system of the adult bee. The three castes can be achieved. Microsporidia are unicellular eukaryotes exposed to fungi. They are obligate intracellular parasites on many known species, most are parasites of fish and arthropods (Delbace, 2009). In 1909, Zander Enoch describes the causative germ *Nosema* first (protozoan) *Nosemaapis*: obligate intracellular parasite, whose cycle takes place in the cell of the bee. During its life cycle, *Nosema* can be in two forms. In the vegetative stage, the parasite reproduces in the body of the bee and the stage of spore, passive and infectious form responsible for the transmission of the disease. More recently, another microsporidian, *Nosemaceranae* was identified in Europe. Spores of *Nosema ceranae* are slightly smaller than *Nosemaapis* (Figure 2) (Higes et al., 2006). The causes that promote the development of this pathology are related mainly during the long winters with prolonged confinement of bees inside the hive (Bailey, 1981). Other factors can also contribute to the development of the disease as improper installation of settlements in wetlands deposited directly on the ground. According to a study in South Africa (Swart, 2003), the highest incidence of the disease occurs in forested areas because of the lack of direct sunlight on the colonies placed in these wooded areas, which could harm the proper regulation of heat and moisture inside the nests and choke the settlements. Improper artificial nourishment given to bees also promotes the development of pathology (Kleinschmidt and Kandos, 1976). Heavily infected bees cannot properly digest their food since the epithelial cells of the intestine were damaged by *Nosema*. The result is a form of diarrhea in the bee, which can then defecate in the hive or on the flight shelf (Bailey, 1954). Then there will be a more or less soiling of the hive. These soils contain millions of spores that become a source of contamination for bees busy cleaning (Bailey, 1955). This pathology causes a weakening of the colonies and an increase in the number of bees: the colony dies with strong reserves of honey and pollen.

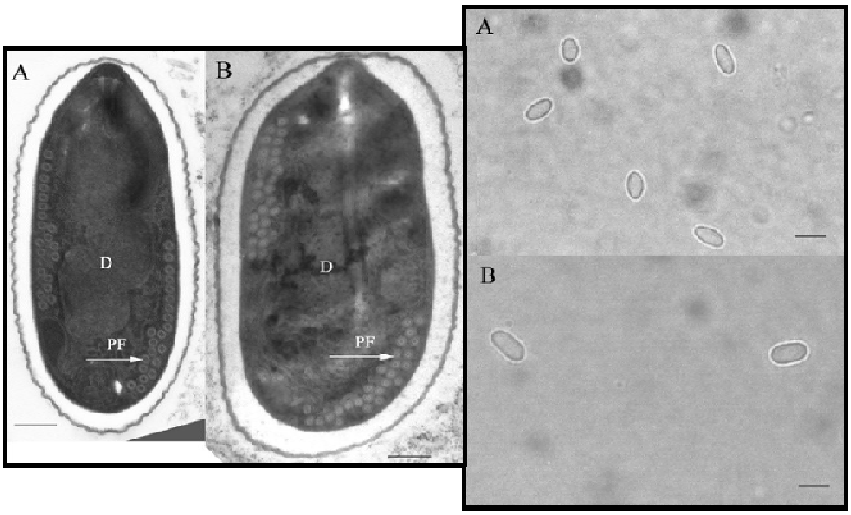

Figure 2: Nosema cerana (A) A dapis (B) observed with electron microscopy and light microscopy (Higeset al., 2006).

Nosema also causes a change in the posterior part of the abdomen (Bailey, 1968). In patients bees, it is whitish, dilated and presents no constriction usual, while in healthy bees, it is yellow to reddish hue and marked constriction. The infection caused by *Nosemacerana* is different from that induced by *Nosemaapis*. *Nosemacerana* disturb bees in several ways: energy stress, decreased lifetime (Fries, 2010; Mayack and Cloudy, 2010) and flight capacity (Kralj and Fuchs, 2010; Adjlane et al., 2012b), disturbance of foraging behavior (Dussaubat et al, 2010;. Higes et al, 2010).In prevention, we must aim to create optimal conditions for the sound development of the whole colony during the season. The only treatment is antibiotics Fumidil

(fumagiline), this antibiotic is mixed with sugar syrup and distributed to the colony. Laboratory experiments conducted in Belgium suggest that acidified food causes the decline in development *Nosemaapis* in the intestine (Mottoul 1996).

4. AMERICAN FOULBROOD

The bacterium *Paenibacillus larvae* subsp. larvae arethe causative agent of American foulbrood, the bacterium has the form of a right stick or slightly curved from 1.5 to 6 cm long and 0.5 cm wide. (Alippi et al., 2004). Spores of thisbacteria ingested arrive in the gastrointestinal tract of a bee larva germinate in the gut after 12 hours (Yue et al., 2008). After the destruction of the tissues, the bacteria cross the intestinal wall and multiply in the hemolymph causing septicemia and the death of the larva. The larvae are most vulnerable to infection in the first larval stages, that is to say 12-36 hours after hatching (Brodsgaard et al, 2000; Genersch et al, 2005). Adult bees are not infected through ingestion of spores of the bacterium (Wilson, 1971).

Symptoms of the disease are seen on the capped brood whose lids are collapsed and breakthroughs. The dead larvae it contains are shooting or dried form of scales and it gives off a strong smell of ammonia. Faucon (1992) reports the presence in the interior of cells of the brood brown scales dark to black as a flat tongue. Larvae and nymphs infected with American foulbrood denature and with bacteria, form an elastic product that stretches when introducing a small toothpick into the affected cell (Prost and Le Conte, 2005). Techniques include microbiological characterization, biochemical and polymerase chain reaction (PCR) (Haynes, 1972; Neuendorf et al., 2004). Diagnosis of AFB is based solely on the identification of the pathogen. Identification methods require a preliminary step of culture, while others can be performed directly on the samples (Murray and Aronstein, 2008; Adjlane et al., 2014; Pellegrini and al., 2017).

To protect his apiary of this pathology, the beekeeper must prevent contamination from already infected apiaries and must also select high vitality colonies. Maintaining a high level of hygiene in the conduct of the apiary is also an important prevention measure. Vis-à-vis resistance AFB is hereditary and answers artificial selection of bees with intense hygienic behavior. Hygienic behavior is defined and Spivak and Reuter (2001) as the ability of bees to recognize, uncapping and removing dead or diseased larvae or nymphs. Antibiotics stop the growth of bacteria or even kill if action during the active life of these bacteria, but antibiotics has no effect on spores. Several antibiotics can be used in the fight against American foulbrood as streptomycin, tetracycline and Terramycin.

But for several years, strains of *Paenibacillus larvae* resistant to these antibiotics have emerged in many parts of the world (Miyagi et al., 1999; Alippi 2000; Mussen, 2000; Evans, 2003). The uncontrolled use of antibiotics poses a risk on bee products through the presence of residues (Lodesani and Costa, 2005; Martel et al., 2006)

5. EUROPEAN FOULBROOD

European foulbrood is an infectious and contagious disease of bee brood less dangerous than American foulbrood (Alippi, 1999). The main causal agent is a bacterium: Melissococcus pluton. Other germs develop secondarily (Lactobacillus eurydice, *Paenibacillus alvei*, *Paenibacillus apiarius*, *Enterococcus faecalis*) (Bailey, 1963 Bailey and Collins, 1982; Alippi 1991). The three castes of bees are affected by the disease. Melissococcus pluton affects the brood, mainly before capping. Encapsulated forms of the bacteria are ingested by young larvae with food (Erler and al., 2017). They thrive in the midgut in their vegetative form and multiply there in droves. Secondary germs penetrate and destroy the larva. Older larvae of more than 2 days are difficult contaminable and adult bees are resistant (Bailey and Ball, 1991). The degree of mortality of larvae measured in an experiment is directly related to the amount of bacteria present in the alveoli. The larvae are less resistant when the number of bacteria is very important (Mcklee et al. 2004).

The pathogen *Melissococcuspluton* does not produce spores, but it can retain its virulence for a year. Adult bees eliminate some of the sick or dead larvae, but few managing to survive diseased larvae will empty their gut and infected cells (Bailey, 1985). The spread of the disease between the colonies and among the apiaries is linked to several factors such as looting, drift, movement of infested colonies and trade of contaminated supplies frames (Forsgren et al., 2005). During infection, the larvae take on a yellowish tint and rotate on the bottom wall of the cell, the back facing the opening. Healthy larvae, meanwhile, are white and fill the whole cell. A typical tangy smell emanates from diseased brood. According to the bee cleaning activity, the sick larvae are removed from the brood, and it follows a brood inadequate (Mckee et al., 2003). The larvae of bees European foulbrood sufferers die 1 or 2 days before the capping of cells, sometimes just after, but always before metamorphosis into a chrysalis (Bailey, 1960). The appearance of the disease is favored mainly by a dietary deficiency of pollen and bad weather can cause this deficiency. A weakened colony with *Varroa* or another cause is also very sensitive to the development of pathology (Delaplane, 1998). The measures relating to sanitation and the prevention of the apiary are the same as for American foulbrood. Chemical treatment is done with antibiotics such as tylosin and oxytetracycline Terramycin (Hitchocok et al, 1970. Thompson and Brown, 2001; Ruth et al., 2003; Waite et al., 2003). These are currently banned for years in European countries. The destruction of the colony, materials and frames of cleaning are mandatory for beekeepers (Belloy et al., 2007).

6. VARROASIS

Varroosis is a parasitic disease of adult bees and brood, due to an external bloodsucking parasitic mite, *Varroadestructor* Anderson and Trueman, 2000 (Figure 3). The *Varroa* lifecycle is strictly linked to that of the bees. It has two phases: phoretic on adult bee, and in the reproductive cells of capped brood of males and workers (Fries, 2005). The *Varroa* reproductive phase lasts for capping the emergence of the bee. The female founder called *Varroa* enters a brood cell just hours before the capping and immerses himself in the larval food (Ifantidis 1988). After capping, it perforates the integument of the nymph creating a nourishment site, boosting its oogenesis and begins her laying. The first egg haploid, will give a male, the other will diploid females through the following stages: egg, larva, and protonymph deutonymph. Mating takes place in the cell, in the fecal accumulation zone. When the adult bee emerges, the founder female and mature females girls out of the cell while the male dies with immature (Faucon 2003). The phoretic phase corresponds to the period between the output of *Varroa* the cell and enters another cell (Martin, 2003). The clinical symptoms of *varroa* include disorders and brood bees (Charriere et al., 2012). The presence of irregular or incomplete brood with dead nymphs atrophied under the cover is one of the main signs of the disease. On adult bees, the symptoms are mainly related to the presence of workers with deformed wings, and that dragged dead bees (Figure 4). *Varroa* spreads through several channels, a bee to bee, from one hive to hive, and even an apiary to another. This is due to several factors, either natural drift by bees, swarming and looting or by transhumance beekeeping and exchanges between beekeepers (Anderson, 1988). Parasitism *Varroadestructor* affects adult bees and brood on three actions: predatory, mechanical and carrier. Repeated hemolymph taken by *Varroa* lead to a decrease in its total volume and its protein levels, undermining the development of the nymph (Bowen-Walker et al., 1999). The decrease in total protein fluctuates between 10 and 50% of parasitized pupae (Dandeu et al., 1991). Yang and Cox-Foster (2005) clearly show that the *Varroa* weakens the immune system of bees and makes it more susceptible to viral and bacterial infections.

The presence of the parasite in the adult bee alters its behavior to the detriment of their regular duties (Faucon, 2003). Parasitism causes deformities and weakness of the young workers. Heavy infestation causes death before the emergence of nymphs and the birth of mutilated bees (Boecking and Genersch, 2008). *Varroa* also causes a drop in weight by about 30% and decreased life expectancy (Bowen- Walker and Gun, 2001). According to Schneider and Drescher (1987), the survival rate of adult bees beyond 25 days, under laboratory

conditions, is about 50% if the bees come from healthy larvae, but is reduced 25% if the larvae are contaminated with three *Varroa (*Nazzi and Le Conte, 2016). De Jong et al. (1982) reported that 6% of emerging parasitized bees have a shortening of the abdomen and localized deformations especially on the wings. In terms of internal organs, a 10% reduction in the size of the acini of hypopharyngeal glands is observed in parasitized bees born (Drescher and Schneider, 1987).

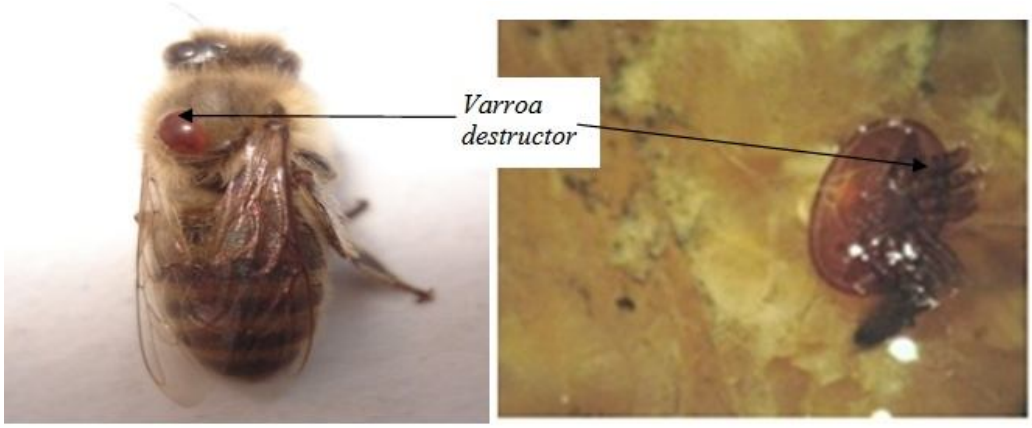

Figure 3: *Varroa destructor* mite parasite of the honey bee

Figure 4: A black bee with wings spread and deformed

The role of mites in the transmission and pathogenesis of some viruses appears twice. On the one hand, by its role *Varroa* vector injected viruses, it carries directly into the haemolymph of the bees. On the other hand, an activating role through the *varroa* bite allows the activation of certain viruses present in a latent state in the bee hemolymph (Tentcheva et al. 2004; Wilfertetal., 2016; Locke et al., 2017). Different factors combine and increase their deleterious effects. The bee, once parasitized by a mite infested with a virus, could indeed be more sensitive to the toxic effects of pesticides in the environment. The fight against *varroa* infestation aims to keep below the damage threshold. Beekeepers have several means of chemical control, biotechnical and natural.

The resistance phenomenon more chemical molecules has been reported by several authors (Lodesani et al,. 1995; Vandame et al, .1995; Londzin and Sledzinky, 1996; Elzen et al, 1988;. Mozes et al. 2000; and Milani Della Vedova 2002; Garcia-Salinas et al, 2006 ; Adjlane et al., 2013a, b).. Forcing beekeepers to move towards the natural control based primarily using oxalic acid, formic acid and thymol. Nevertheless, control of *varroa* falls on the diaper is important and determines the period and the treatment mode.

7. VIRUSES

Viruses are obligate intracellular parasites. They use the cellular machinery of the host to replicate infested and thereby cause damage. They are composed solely of nucleic acid surrounded by a protein capsid. In the bee, about 18 viruses are known to date. With the exception of filamentous virus (FV, filamentous virus) that is considered a DNA virus genome, all the honeybee viruses identified to date are small virus positive single-stranded RNA and non-enveloped capsid. The bee virus belongs to the family of dicistroviridae (Olivier and Ribiere, 2006). They are round and are 15 to 30 nm in diameter (Dainat et al., 2008). Horizontal transmission between individuals of the same generation can be direct or indirect. This can arise, for example direct contact with skin. Bees can transmit the virus to chronic paralysis (CBPV) by contact, particularly in terms of injuries (Blanchard et al., (2007). Contamination is called indirect when is via an intermediary. The viral particles excreted via bee excrement affected by CBPV can contaminate individuals in contact therewith (RIBIERE et al., 2000). The transmission of the virus can also be vertical. A queen contaminated with a virus identified in the ovaries and spermathecal can lay infected eggs (CHEN et al., 2006).

The DWV (Deformed Wing Virus) was originally isolated from the adult bees in Japan on infested colonies *Varroadestructor* (BALL, 1985). He was later identified as a cause of mortality of bee colonies in many countries (Ball, 1985; Ball, 1987; Bailey and BALL, 1991; Meana and al., 2017; Brettell and al., 2017). It is the most prevalent viruses and dangerous now (Kajobe et al, 2010;. Mockel et al, 2011;. Hongxia et al, 2012; Adjlane et Haddad, 2014). The name given to the virus comes from the characteristic symptom of the deformed wings or poorly developed in newly hatched bees from infected colonies (Ball, 1993). Deformed wing virus affects the eggs, larvae, pupae and adult bees (Allen and Ball, 1996). The same virus can be detected in all parts of the body of the bee (Chen et al., 2004; Yue Genersch and 2005). The infected nurses transmit the virus to young larvae through the larval jelly (Ball, 1987). Adult bees transmit the virus during the trophallaxis (Bowen-Walker et al., 1999; Norstrom et al., 1999)

The ABPV (Acute Bee Paralysis Virus) was discovered in laboratory work on the identification of the causative agent of the viral disease CBPV (Bailey et al., 1963). Before the spread of the mite Varroa destructor, this virus has never been associated with a death or illness in the colony (Bailey and Gibbs, 1964; Bailey et al., 1981). It was detected in the brain (Bailey and Milner, 1969), in the tissue and salivary glands of bees (Bailey, 1965). In Europe, large amounts of virus were detected on adult bees and brood dead colonies heavily infested with *Varroadestructor* (Ball, 1985; Carpana et al, 1990; Falcon et al, 1992).

The BQCV (Black Queen Cell Virus) was isolated for the first time on a larva of a dead queen found partially decomposed in a dark cell with a royal black color of the wall, hence the name of this virus (Bailey and Woods, 1977). BQCV is associated with the parasite *Nosemaapis* in its development cycle. The peak of infection with this virus is in spring and early summer (Bailey et al., 1983). This virus is rarely detected on the worker larvae, according to Bailey et al (1983) the brood of workers receive less food than the larva of the queen, so if there is a viral infection of the amount ingested is less important than that ingested by the queen. The BQCV can be transmitted vertically, Chen et al (2006) showed the presence of this virus in the intestine and the ovaries of queens (Chen et al., 2006). Its transmission is largely independent of the mite *Varroadestructor*. In a study conducted by Tentcheva et al (2004), the BQCV was

most frequently detected in adult bees in the brood. Still, according to the same authors, this virus does not cause visible symptoms of infection in adult bees.

The SBV (Sac Brood Virus) is an infectious disease of the bee causing huge losses for the colony. The characteristic symptoms are sparse brood and larvae in the form of small bags. This is the first bee disease attributed to a virus, the virus was identified in a diseased brood in 1917 by WHITE (Bailey, 1969). Then this same virus was isolated by Bailey et al (1964). Although primarily a disease of the larvae. The SBV multiplies in the adult bees without causing obvious symptoms (Bailey, 1968). It is much more contained in the head of the adult bees infected than in other parts of the body and large quantities of virus were detected in the feeder glands (Mussen and Furgala, 1977). This virus is transmitted to young larvae by the nurse with the larval jelly (Hitchcock, 1966).

The KBV (Kashmir Bee Virus) was discovered in 1974 on the Asian honey bee *Apis cerana* from the Kashmir region (Bailey and Woods, 1977). The KBV does not cause clinical symptoms characteristic (Hornitzky, 1987). This virus can be detected on adult bees and brood (Bailey et al. 1979; Stolz et al., 1995). *Varroadestructor* can transmit the virus between adult bees and between nymphs (Shimunuki and Hung, 1999; Hung et al., 2000; Chen et al., 2004; Kalashnikov and Udina,2017). Tentcheva et al (2004) studied the prevalence and seasonality of six bee viruses in healthy colonies in 36 apiaries throughout France. Overall, KBV was less frequently detected. The same virus was also detected on *Varroa* mites. The impact of the infection on virus KBV bee colonies and its role in the mortality of colonies infested with *Varroa* are poorly understood.

The causative agent of CBPV (Chronic Bee Paralysis Virus) was isolated in 1963 by Bailey et al. Bees affected by the virus become unable to fly, trembling, creeping, crammed into the hives, the dislocated wings with swollen abdomen (Rinderer and Rothenbühler, 1976; Bailey and Ball, 1991). The virus input channel can be oral or dermal (Ball, 2004; Chen et al., 2006). The CBPV easily passed between adult bees experimentally by application to the surface of the cuticle, the virus invades the body of adult bees via the epidermal cytoplasm (Bailey et al., 1983). Infection with less effective orally, can also contribute to the spread of the virus by sharing food (Bailey, 1965). This virus can be detected with the queen, and in all stages of development of the bee (Chen et al., 2005). When diagnosing the virus must take into account the amount of pathogens, themultiplication of the virus, host susceptibility and the stage of development of the latter. Currently, the technique of RT-PCR or polymerase chain reaction to amplify the genetic material, after the action of a reverse transcriptase to convert RNA into DNA is most commonly used (Gauthier et al., 2007; Rachel et al. , 2011; Hongxia et al, 2012). It not only allows a certain identification of the virus, but a dosage of viral material (Berthroud et al., 2005).

8. CONCLUSION

The establishment of effective methods of treatment against apiolary pathologies requires a precise knowledge of the biology and development of these pathologies in order to improve the methods of treatment by beekeepers. This literature review shows the side effects of these diseases on bee colonies, mortalities recorded in recent years are caused mainly by the bee diseases. Considerable efforts and collaboration between researchers and beekeepers to improve the health of bee colonies and decreased losses worldwide

Author's contribution: All authors contributed equally to this work (The drafting and correction of the article)

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