



E-ISSN: 2717-8633

Sayı(Number) 9, Nisan(April) 2025

DERLEME MAKALESİ/REVIEW ARTICLE

Geliş Tarihi(Receive Date): 03.12.2024

Kabul Tarihi(Accepted Date): 03.01.2025

Bee diseases and treatment methods

Mert Gül^{a,£}, Ebru Halvacı^{b,£}, Hatice Kars^c, Teslime Kozak^d, Damla Ikbali^e, Alper Ozengül^f, Fatih Sen^{g,*}

^a Sen Research Group, Department of Biochemistry, Kütahya Dumlupınar University, Kutahya, Türkiye, ORCID: 0009-0005-7215-541X

^b Sen Research Group, Department of Biochemistry, Kütahya Dumlupınar University, Kutahya, Türkiye ORCID: 0009-0003-6062-7622

^c Sen Research Group, Department of Biochemistry, Kütahya Dumlupınar University, Kutahya, Türkiye ORCID: 0009-0002-1107-0605

^d Sen Research Group, Department of Biochemistry, Kütahya Dumlupınar University, Kutahya, Türkiye ORCID: 0009-0006-9446-8449,

^e Sen Research Group, Department of Biochemistry, Kütahya Dumlupınar University, Kutahya, Türkiye ORCID: 0009-0005-5206-8810

^f Sen Research Group, Department of Biochemistry, Kütahya Dumlupınar University, Kutahya, Türkiye ORCID: 0009-0008-4261-2263

^g Sen Research Group, Department of Biochemistry, Kütahya Dumlupınar University, Kutahya, Türkiye ORCID: 0000-0001-6843-9026

Abstract

Bee diseases are one of the main problems related to beekeeping activities. Harmful microorganisms such as parasites, bacteria, viruses, and fungi cause diseases in honey bees juvenile and adult periods. These disease-causing agents enter the colony by various means and spread rapidly from colony to colony, apiary to apiary, and region to region. Therefore, bee diseases are the main problems that negatively affect beekeeping activities, cause colony losses, and limit production efficiency. In bee diseases, symptoms such as watery stools, thirst, tremors during rest, unusual wing angles, restless behavior of bees in the chronic period, and changes in body physiology are observed. These symptoms appear mostly in the spring. However, the disease suppresses the hive during the summer period. Therefore, to minimize the problems in the hive, there is a need to control the diseases and develop control methods. Colonies should be observed correctly in the spring and fall periods and strengthening activities should be carried out. It is recommended to move sick colonies to disinfected hives after they have been treated. The reason for this is to prepare the colony for the winter period without allowing brood development to stop during the summer period. It is very difficult to say that beekeepers are made aware of this field, the correct treatment methods are applied, and they have access to the relevant resources. It is seen that some existing treatment methods and drugs are not direct solutions for bee diseases. For this reason, this review covers bee diseases and pests that can cause colony losses in detail. These diseases are mainly classified as 'bacterial', 'viral', 'protozoal', and 'other colony pests'. In addition, traditional and new technological treatment methods for bee diseases are also briefly mentioned.

© 2023 DPU All rights reserved.

Keywords: Bacterial Diseases; Bee Diseases; Medication Therapy; Pest Control

*Corresponding author: fatihsen1980@gmail.com

£ These authors equally contributed to this study.

Arı hastalıkları ve tedavi yöntemleri

Öz

Arı hastalıkları, arıcılık faaliyetleri ile ilgili temel sorunlardan biridir. Parazitler, bakteriler, virüsler ve mantarlar gibi zararlı mikroorganizmalar bal arılarının yavru ve ergin dönemlerinde hastalıklara neden olmaktadır. Bu hastalık etmenleri çeşitli yollarla koloniye girmekte ve koloniden koloniye, arılıktan arılığa ve bölgeden bölgeye hızla yayılmaktadır. Bu nedenle arı hastalıkları, arıcılık faaliyetlerini olumsuz etkileyen, koloni kayıplarına neden olan ve üretim verimliliğini sınırlayan başlıca sorunlardır. Arı hastalıklarında sulu dışkı, susuzluk, dinlenme sırasında titreme, alışılmadık kanat açları, kronik dönemde arıların huzursuz davranışları ve vücut fizyolojisinde değişiklikler gibi belirtiler görülmektedir. Bu belirtiler çoğunlukla ilkbaharda ortaya çıkmaktadır. Ancak hastalık yaz döneminde kovani baskı altına almaktadır. Bu nedenle kovandaki sorunları en aza indirmek için hastalıkların kontrol altına alınması ve mücadele yöntemlerinin geliştirilmesine ihtiyaç vardır. İlkbahar ve sonbahar dönemlerinde koloniler doğru gözlemlenmeli ve güçlendirme çalışmaları yapılmalıdır. Hasta kolonilerin tedavi edildikten sonra dezenfekte edilmiş kovanlara taşınması önerilmektedir. Bunun nedeni, yaz döneminde kuluçka gelişiminin durmasına izin vermeden koloniyi kış dönemine hazırlamaktır. Arıcıların bu alanda bilinçlendirildiğini, doğru tedavi yöntemlerinin uygulandığını ve ilgili kaynaklara ulaşabildiklerini söylemek çok zordur. Mevcut bazı tedavi yöntemleri ve ilaçların arı hastalıkları için doğrudan çözüm olmadığı görülmektedir. Bu nedenle bu derlemede koloni kayıplarına neden olabilen arı hastalık ve zararlıları detaylı olarak ele alınmıştır. Bu hastalıklar temel olarak 'bakteriyel', 'viral', 'protozoal' ve 'diğer koloni zararlıları' olarak sınıflandırılmıştır. Ayrıca, arı hastalıkları için geleneksel ve yeni teknolojik tedavi yöntemlerinden de kısaca bahsedilmektedir.

Anahtar Kelimeler: Bakteriyel Hastalıklar; Arı Hastalıkları; İlaç Tedavisi; Haşere Kontrolü

1. Introduction

Bees are defined as beneficial insects whose first human existence dates back to the Cretaceous period in the Mesopotamian era. The most important products of bees are honey, beeswax, royal jelly, royal jelly, bee venom, pollen, and propolis, natural products that directly affect human health [1]. In addition, the pollination factor that bees provide to plants in their natural habitats is vital for the protection of ecological balance and the continuation of agricultural production. For this reason, beekeeping has been seen as a source of income or healthy nutrition for the rural population from past to present. Today, with the transition from traditional production to modern methods in beekeeping, there is a rapid increase in the number of bee colonies. With this increase, problems such as buying and selling of bees between countries, disruptions in queen production, lack of control in the sale of queen bees, increase in itinerant beekeeping, shrinkage of beekeeping areas, widespread use of pesticides arise in the beekeeping sector [2], [3]. Bee diseases, colony losses, and pesticide problems threaten all living things in and around the hive. Similarly, the trade of bees, bee products, and beekeeping materials between countries causes bee diseases to spread in a short time. Bee diseases are seen especially in the spring months [4]. The reason for this is that in the spring months, the queen bee's activity of raising offspring increases greatly with the laying period and it is faced with predatory insects with spring rains. During this period, it is necessary to be very sensitive to bee diseases, to prevent the colony from catching cold during hive controls, and to sterilization of the hive during and after harvest. It is very important to protect the colony from disease by considering the risks of transmission from other colonies such as preventive measures, fight against pesticides, and regular hive maintenance [5]. Bee diseases are classified as bacterial (American and European Foulbrood, Septicemia, Crusted Larvae Disease), marketer (Varroa Jacobsoni and Acarapis Voodi), viral (Chronic and Acute Bee Paralysis), fungal (Lime and Stone Disease) and protozoan (Nosema and Amoeba) according to the factors causing the disease [6], [7], [8]. In addition, Adult and Fledgling Bee Diseases are also seen depending on the host of the disease. Many pathogens, pests, and insects cause diseases in both the developmental and adult stages of bees. However, not all of them are equally dangerous, and some diseases cause the hive to be destroyed within a few weeks [9]. For example, bee diseases classified as very dangerous, such as American foulbrood, Lime Disease, and Varroa, spread rapidly throughout the colony and require national beekeepers association control programs at very advanced stages [10]. American foulbrood disease causes the bee eggs in the larval stage to turn yellow and even brown in the later stages. The most prominent feature of a hive infected with the disease is that the dead brood is not easily cleaned from the comb cells [11]. In this case, the queen will not lay eggs until the honeycombs

are cleaned, so the offspring are not organized but scattered [12]. The bees that die from the disease are seen in the honeycomb in a watery, soft mushy consistency. The dead larvae are watery and sticky and cover the base of the eye. When a stick is inserted and removed from the honeycomb, it grows up to 6-8 cm and infects other honeycombs. For this reason, it is very difficult to remove and clean the closed-eye bases [13], [14]. Another example is Varroa disease, the parasitic insects in the closed eye suck the blood of the bee in the larval, pupal, and adult stages, reducing the working activity in the hive [15]. In addition, the larval remains dry out over time and adheres to the base of the eye as a host carrying millions of spores. However, if the pupa dies in the pupal stage, their tongues harden and pierce the honeycomb. The most prominent feature of Varroa disease is the appearance of wingless bees in the hive. Bees are exposed to many disease agents, ecological disorders, and insecticides during the development period [16], [17], [18]. In this case, the rate of development of the diseased hive decreases, and the number of adult bees and the amount of offspring are visibly reduced. There is a decrease in the activity of bees to collect pollen and nectar for the hive. Therefore, bee diseases cause great losses in bee-keeping countries [19], [20]. However, it is very difficult to say that a conscious treatment method is applied for bee diseases. Beekeepers should be made aware of issues such as unconscious use of antibiotics, not paying attention to hive hygiene, not performing spring maintenance, not cleaning the hive after harvest, direct transmission between parasite colonies, plundering of weak colonies, contaminated honey contaminating healthy colonies [21], [22].

Within the scope of this review, the creation of a resource where beekeepers can get detailed information, about bee diseases, disease-causing factors, and treatment methods from the past to the present are mentioned. Diagnosis of bee disease, disease symptoms, according to the factors causing the disease; bacterial, viral, protozoal, fungal, parasitic, or honey bee pests are discussed in detail with literature research [23].

2. Bacterial diseases

2.1. American foulbrood rot

American foulbrood disease causes disease in honey bees *Apis mellifera* and other *Apis* species [24]. It is seen in honey bee larvae and is a highly contagious bacterial disease [25]. When infected, it kills entire colonies [26]. The causative pathogen of the disease is known as *Paenibacillus larvae subsp. larvae* [27]. Apart from infecting beehives kept in the same region, it can also infect beehives kept in different regions around it. *Paenibacillus larvae* spores can contain 2.5 billion spores in the infected larva [28]. *Paenibacillus larvae subsp. larvae* spores remain viable for 33 years in the hive, 60 years in the soil, and 45 years in the foundation comb [29]. Spores that can remain viable for many years cause disease in the following years [30]. The structure of *Paenibacillus larvae* bacteria is straight or curved rod-shaped, 1.5-6 µm in length and 0.5-0.8 µm in width. Gram-positive [31] and microaerophilic, *Paenibacillus larvae* are found in nature in the form of single chains or filaments [32]. *Paenibacillus larvae* have two forms, spore and vegetative. The vegetative form is not dangerous for bees, only the spore form causes infection in bees [33]. *Paenibacillus larvae subsp. larvae* spores are ellipsoid-shaped. Bacterial spores are resistant to chemicals and temperature changes. *Paenibacillus larvae* spores are highly resistant to external conditions. Even in the dry remains of larvae that died from this disease, viable spores were found 69 years later. Spores can survive for more than 40 years in the comb, 1-10 years in honey, and 5 days in molten beeswax. For this reason, sterilization of beeswax at high temperatures is important. American foulbrood shows its effect depending on the age of the larvae. When the age of the larva exceeds 32 hours, American foulbrood cannot show its full effect and its sensitivity decreases. After the larva reaches 53 hours of age, American foulbrood is no longer a threat. For larvae less than 24 hours old, 1 *Paenibacillus larvae* spore is sufficient for infection. *Paenibacillus larvae* spores begin to reproduce 24 hours after the larva has ingested food. This bacterium reproduces in the middle intestine of the bee and enters the body cavity by crossing the intestinal wall [34]. Approximately 2.5 million spores grow in the body of the infected larva. American

foulbrood disease is transmitted from colony to colony through theft and hive straying. The exchange of frames with young between diseased colonies and healthy colonies, honey and pollen from diseased colonies, and swarming are important factors in the spread of the disease [35]. American foulbrood bacteria can be easily transported by queens, unsterilized combs, beekeeping tools and clothes, beekeepers, combs, and swarming bees [36].]. Another factor in the spread of the disease is the improper sterilization and use of diseased old combs. The disease can be seen in worker bees, drones, and queens in the larval stage. However, the disease is rarely seen in adult drones and queens. In strong colonies, the disease is very difficult to recognize. The progression of the disease is understood by the increase in bee losses. After increasing bee losses, the vitality in the flight hole decreases and the bees wander involuntarily on the flight board. It is an important issue that worker bees throw broods out of the hive. This should not be confused with the action of starving bees to protect the colony, because when American foulbrood starts in the hive, the bees consume large amounts of honey and run out of honey. To cope with this situation, they sacrifice their young and expel them from the hive. Larvae born in a healthy hive have a pearly white, shiny color. At the base of the comb chamber, the larva is in a "C" shape. When infected, this structure disappears fills the cell, and dies upwards. Larvae that die from American foulbrood disease turn dull-white, light brown, dark brown, and black in color. The structure of larvae that die from American foulbrood is initially watery and slightly sticky. As the disease progresses, the larvae become like glue, and their color turns brown [37]. There is also a glue odor in the combs. The larvae that turn brown are subjected to the "larval elongation test" by pulling them to the right and left with the help of a suitable tool. When elongation between 2.5 cm and 10 cm is observed, it is considered as a marker of American foulbrood (figure 1). It is also possible to identify the disease with the stick test, which is 98% accurate. In the stick test, diseased larvae are placed in a bottle and diluted. Then a drop of sample is taken and placed on the preparation. When a color change is observed, the presence of the disease is confirmed. In the honeycombs with the disease, the pupal eyes have an asymmetrical appearance. There is a mixed color appearance in the honeycomb eyes. The dead larvae in the combs lie on the bottom surface and are very firmly adhered to each other. When the larvae are dried, they are hard and very difficult to clean. The membranes of the pupal eyes are withered, collapsed inwards and have holes the size of the ball of a pin. Juveniles that die during the larval stage are found in closed eyes. This is the main difference between American foulbrood and European foulbrood. The larvae that are dead in the closed cavity die upright in the comb cavity, but this is not observed in European foulbrood [38].



Fig 1. American foulbrood disease colony with visible drifting fry lava (Reprinted with permission from [39], Copyright MDPI).

One of the main methods of combating American foulbrood is to keep the apiaries clean and tidy. Pieces of honeycomb and propolis should not be thrown around the combs. Diseased combs should not be used again and should be destroyed by burning. Bees should be taken to places where pollen and nectar are high. Strong bee colonies should be used and queen bees should be replaced if they are inefficient. Beekeeping activities should be carried out at a distance of about 6 km from other apiaries. When purchasing a colony, the colony should be checked for diseases. Beeswax should be sterilized at 120°C for 15 minutes before being made into combs. Hives should be positioned in a U or S shape. Hives should be checked frequently and diseased colonies and all beekeeping products should be destroyed as soon as the disease is detected. Beekeeping equipment should be more than one. Potassium hypochlorite, lye, kefir, hydrogen peroxide, and chloramine potassium hypochlorite can be used when disinfecting beekeeping materials. Antibiotics containing oxytetracycline hydrochloride (OTC) and sodium sulfathiazole should be used in the treatment phase of the disease. However, antibiotics are not effective in sporulated forms but in vegetative forms [40].

2.2. European foulbrood rot

It is an infectious and bacterial disease seen in most parts of the world. It affects honey bee brood. The causative pathogen of European foulbrood disease is non-spore forming, gram (+) *Melissococcus plutonis* [41]. This disease can also be caused by other secondary microorganisms. These microorganisms include *Streptococcus faecalis*, *Bacillus eurydice*, *Paenibacillus alvei*, *Bacillus cereus*, *Bacillus laterosporus*, *Bacillus subtilis*, *Bacillus orpheus* and *Corynebacterium pyogenes* [42]. Secondary bacteria are effective on the odor and consistency of dead larvae, they do not affect the occurrence of the disease. This disease is mostly seen in weak hives in spring and early summer (usually in May and June), when brood rearing is intense [43]. In addition, this disease is particularly common when nectar is scarce. The number of young bees is directly related to food production. To produce large amounts of food, young bees must be numerous. In this way, enough food is produced for the larvae. It shows its effect more in the larval stage of bees. The disease is more effective on 3-4 day old larvae. Carrier adult bees are less affected by the disease [44]. The pathogen of this disease is transmitted to the larvae by the worker bees during the feeding of the larvae with the food taken by mouth and is rapidly transmitted. In this way, the bacteria settle in the digestive system and develop in the middle intestine. In the meantime, since they feed on the food in the intestine of the larva, the larva loses nutrients and cannot develop because it rapidly consumes the contents of the intestine. After infection, death is usually seen after 4 days, and death is seen before the larva passes to the pupal stage [45]. After the larva has passed into the pupal stage, it is excreted into the comb chamber with feces. The disease is also transmitted to healthy larvae by the contact of worker bees while cleaning the residues in the comb cells. The causative agent of the disease survives for 3 years in the intestine of the bee, 1 year in old combs, 15-20 minutes in boiling water, 65 days in beeswax, and 2 minutes at 116°C. Symptoms of the disease are seen in climatic conditions such as poor and weak hives in winter, prolonged cold and humid weather in spring, malnutrition, stress, and lack of nectar and pollen. Combining weak and diseased colonies with healthy colonies, using unsterilized tools that were previously used in the infected hive can also cause the disease. If there is an excess of young bees, the hive produces the necessary food for the larvae. If the hive receives a large amount of nectar, the severity of the disease decreases to the same extent and the disease begins to recover when the colony conditions return to normal [29]. The larvae carrying the disease are usually detected and removed from the hive before they show signs of the disease. Therefore, the disease can go undetected for long periods. Most brood deaths occur before the honeycomb eyes close, so the disease occurs in open honeycomb eyes, but if the disease is severe and advanced, deaths can also occur in closed eyes. If death occurs in closed eyes, the eye will collapse, perforate, and discolor. The bees that die from the disease are curled into the letter "C" at the base of the comb eye. This is the most important symptom in diagnosing the disease. They do not adhere to the honeycomb, they can be easily removed from the honeycomb with the help of a match. The color of dead larvae first changes to white, then brown, and then black [46]. When the hive lid is opened, the odor perceived due to the dead larvae inside the hive is the smell of spoiled meat/fish or sour brewer's yeast. Another symptom is that dead larvae can be easily removed from the comb cavity without elongation when the dead larvae are pulled out with the garbage [47]. The consistency of the

dead larvae is soft and watery at first and then hardens to a pasty consistency. Colonies infected with the disease show reluctance and weakness. Larvae that have died but which the worker larvae cannot expel rot. The decaying larvae have little or no stickiness. With the death of the larvae, the proliferation of the causative agent stops. To diagnose the disease, an easy milk test can be performed. Healthy or sick larvae can be identified according to their milk coagulation time. Healthy larvae coagulate milk in around 13 minutes. Sick larvae coagulate milk in 2 minutes. Early diagnosis plays an important role in the treatment of the disease. When the disease is not diagnosed and intervened in time, the disease progresses. Except in severe cases, the destruction of bees and combs is not necessary. If the disease progresses, the hive may need to be burned [43]. As a way of controlling and preventing the disease, colonies harboring the disease agent should not be merged with other colonies and should not be moved. Colonies should be moved to nutrient-rich areas with better pollen and nectar. Nutrition should be provided with syrups containing minerals and vitamins. It should be ensured that the queen bee does not leave the hive to prevent the formation of new larvae and that the available nutrients are sufficient for the larvae in the hive. The queen in the colony is prevented from laying eggs by keeping her inside the hive for a while. In hives with the disease, the queen should be renewed and young queens should be used. Bees are kept away from stress. The diseased combs in the hive should be removed from the hive. Honeycombs with strong and healthy brood from other hives should be taken into the hive. The equipment and tools used must be sterile. The disease can be treated with antibiotics such as oxytetracycline, erythromycin, etc. However, the use of any antibiotic for the treatment of this disease is prohibited in Turkey. An antibiogram test is used to determine the antibiotic that will be effective. In the use of antibiotics, a specialist should be consulted and applications should be made accordingly. Because each product is a substance that must be taken at certain intervals and in certain doses. If antibiotic administration is not done carefully, the bee colony and the quality of the honey will be damaged. Antibiotics used unconsciously increase the resistance of bacteria and cause residue in honey. Honey from the hive that has been given antibiotics should not be consumed for a long time. Even when the larvae recover from the disease, the disease agent continues to live in the honeycombs in the hive and the disease may recur. European foulbrood is a disease that can be encountered quite frequently. But in some cases, when the infection level is low or the colony is strong, the bees can cope with the disease.

2.3. Septicemia disease

Septicemia is also known as blood poisoning [48]. It is a fatal disease caused by a bacterium called *Pseudomonas apiseptica* (*Pseudomonas aeruginosa*) [49]. It is a non-spore-forming, motile, and gram (-) bacteria. This causative bacterium is found in plants, swamps, stagnant water, and moist soils. This disease is transmitted through the respiratory system (trachea), from where it passes into the blood fluid and causes infection. The possibility of disease is triggered by stress factors. Unfavorable weather conditions, intensive artificial feeding, the difficult adaptation of swarming bees to the hive they come from, feeding with too dense sugar syrup, and honeycombing stress trigger the disease. The causative agent is found in the hemolymph of bees but rarely causes disease. The hemolymph of healthy bees is yellow. The hemolymph of sick bees varies in color from white to brown. *Pseudomonas aseptica* is found in the gut of young bees that feed on pollen. It is not found in the gut of young bees emerging from the honeycomb. When the bees feed on honey, the bacteria in the gut are destroyed. The death of the bees infected with the disease becomes inevitable in a short time. The death rate is highest 20-36 hours after the bee is infected [50]. Adult bees carrying the disease are seen in pairs or singly. The symptoms of this disease are; that the color of the bees starts to turn black, the ability to fly decreases and then disappears, and there is a rapid loss of reflexes in their muscles. The most important symptom of the disease is the degeneration of the muscles. Since the muscles of diseased bees are deformed, they cannot be held at the joints. For this reason, when the head, chest, abdomen, and legs are touched, these parts are torn from their joints [29]. If the patient becomes unable to defend the bee, there is a risk of being easily caught. Since the muscles cannot fulfill the function they are supposed to do, the bees cannot fly and walk in a dazed state. In a weakened state, they move their mouthparts and legs. Food consumption first decreases, then stops and the colony is weakened. Another symptom is a change in blood color. In healthy bees, the blood color is yellowish or ashen. But the blood color of the sick bee changes from light brown to white. Dead or dying bees in diseased hives have a rotten, sour odor. There is no known cure for the disease. However, some precautions can be taken. These

measures can be taken such as avoiding stress factors, establishing hives in clean, dry, sunlit places, avoiding contaminated water in the environment, and keeping colonies in environments rich in nutrients such as pollen and nectar. When the nutrient sources in the environment are low, vitamin-added sugar syrups should be given. Intensive artificial feeding should be avoided. Work with strong bee colonies. The use of antibiotics is prohibited in this disease.

2.4. Crusted larva disease (Dusty larva disease)

Paenibacillus pulvifaciens bacterium is the causative agent. This disease is rarely encountered. The reason for the rarity is that the disease is rare and beekeepers have difficulty in diagnosis. The dead larvae are crusted specific to the disease. The crusts can have different colors from yellow to brown. They can be seen extending from the bottom to the top of the honeycomb. When the dead larvae are crumbled, they fall as dust. *Paenibacillus pulvifaciens* is 0.3-0.6 x 1.5-3.0 µm in size. It can grow on nutrient agar. It is very similar to *Paenibacillus larvae*, but its spores do not make Brownian motion when tested with the modified drop technique. Another difference is that *Paenibacillus pulvifaciens* can grow in nutrient agar at 20°C [29].

2.5. Spiroplasmosis

Spiroplasma species bacteria cause spiroplasmosis in adult bees. This bacterium is motile, spiral-shaped, and has no cell wall [51]. *Spiroplasma* is 2-10 µm in length and 0.7-1.2 µm in width. It is very easy to visualize with a dark field microscope [52].

3. Viral diseases of bees

3.1. Sackbrood rot (SBV) (Sackbrood)

Also known as bag rot or bag disease. The disease agent is a very small virus called *Morator fistulas*. This disease was first described by White in 1917 [53]. There is no information on whether this disease is seen in our country [54]. It causes disease in workers and drone larvae. It is seen in pupae in the pupal stage [55]. Adult bees become infected due to contact with the fluid containing the virus in the body of the sick larvae while throwing the sick larvae out of the hive. Larvae acquire the virus through worker bees, which are responsible for the development and feeding of the brood. The virus multiplies in the brood food glands of the worker bees and passes to healthy larvae fed by the worker bees [56]. The diseased larvae die after the honeycomb is sealed and during the transition to the pupal stage. The incubation period of the disease is 6-7 days. Larvae are most susceptible to the disease when they are 2 days old. Infected larvae cannot pass into the pupal stage and fluid accumulates around the skin [57]. The head of the infected larva faces the comb chamber and death occurs in the upright position. Mortality is low in open comb cells but high in sealed comb cells. Young larvae infected with this virus show retarded and inadequate development and queen bees show a decrease in egg-laying. Sick colonies become weak and honey yield decreases. This disease is seen in hives that spend the winter in poor conditions and therefore emerge in the spring with weak and neglected hives. The exchange of comb and food between hives also triggers the spread of the disease. It is not as lethal as other brood diseases. Infection decreases and disappears in winter. It is most common in spring and summer [58]. The color change is important as a sign of the disease. The larval color is white at first, then yellow, but turns dark brown when the disease progresses. The color of the dead larva is gray-black. The dead larva dries and crusts [59]. It is hardened and curled sideways in the shape of the letter "L" in the comb chamber [60]. If the honeycomb eyes are opened, the head of the larva can be seen curled up and to the side. The causative virus disrupts the order required for the molting of the diseased larva, so that the old skin, which cannot be separated from the head, cannot be detached. Fluid accumulates between the two skin parts [61]. For this reason, the head swells and curls up, forming a structure similar to a jumpsuit. The larvae look like a bag filled with water. The dead larva can be easily removed from the honeycomb

with the help of a needle. The honeycombs look like a puzzle. Dead larvae have a clear greenish liquid between the larval shirt and the body. Dead larvae have no odor. The dead larvae are removed from the combs by the worker bees and thrown out of the hive. The disease is a brood disease, but the virus that causes the disease also affects adult bees [62]. Since it is a viral disease, it is diagnosed by molecular techniques. The diagnosis of the disease is made by molecular techniques ELISA and RT-PCR. Since the disease is caused by a virus, there is no known drug for its treatment. Some precautions should be taken to prevent the disease. In order to prevent the occurrence of the disease, it is necessary to feed the bees well in the spring, pay attention to the temperature of the hive, nutrient supplementation, not to catch the young cold and cut out the honeycombs in which the disease is detected, change the queen bee, increase the number of worker bees, hives should be located in places without moisture, attention should be paid to moisture intake from the bottom. In order to maintain moisture content, hives should be placed 30-40 cm high.

3.2. Acute bee paralysis

Acute bee paralysis disease is caused by the acute paralysis virus which is in the Dicistroviridae family [63]. Acute paralysis virus is seen in the summer season. If *Varroa destructor* is seen in the colony in addition to acute paralysis virus, deaths are observed in adult bees and larvae. *Varroa destructor* feeds on the blood of bees with the virus, which facilitates the multiplication of the virus [64]. In this way, the acute paralysis virus has a lethal effect. *Varroa virus* facilitates the transmission of acute paralysis virus to adult bees, larvae, and pupae. Acute paralysis virus spreads rapidly in the colony. It targets young larvae and adult bees. It draws attention to rapid adult bee death. Larvae infected with the acute paralysis virus have an appearance similar to American foulbrood or European foulbrood (figure 2).

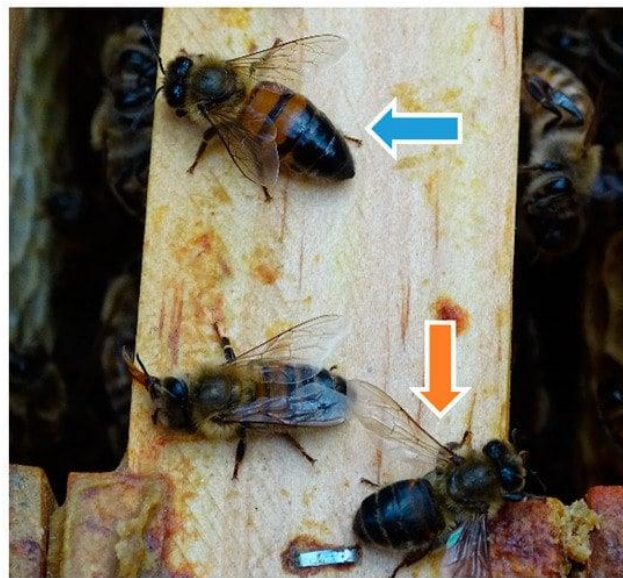


Fig 2. Colony inspection of hairless, dark-colored, short-bellied worker bees and ant-like bees holding their wings at unusual angles (Reprinted with permission from [82], Copyright MDPI).

3.3. Kashmir bee virus

Kashmir bee virus, a member of the *Dicistroviridae* family, was discovered in the Kashmir region of India [56]. This virus was first found in *Apis cerana* bees, and then in *Apis mellifera* bees in Australia. Kashmir bee virus is fatal for

adult bees, larvae, and pupae. Kashmir bee virus is similar to the acute paralysis virus with its disease-causing and spreading characteristics. Kashmir bee virus survives in adult bees and pupae without showing signs of disease. Kashmir bee virus strains have a high ability to produce disease. Very few Kashmir bee viruses are sufficient to cause infection. It multiplies rapidly in the blood of pupae or adult bees and becomes lethal in a short time. This virus becomes much more effective with varroa. Kashmir bee virus combined with varroa destroys colonies [65].

3.4. Chronic bee paralysis

Chronic bee paralysis was first reported in the USA in 1809 in an article written by Huber. Chronic bee paralysis was identified by Burnside in 1933. Burnside brought the disease to light by infecting healthy hives with infected material obtained from infected hives. The paralysis virus is satellitic, 30-65µm in size. The bee's immune system blocks the reproduction of the chronic paralysis virus. Chronic bee paralysis is more common in temperate regions. The disease is recognized by its symptoms. The typical symptom of chronic bee paralysis is the separation of queen and worker bees from the hive. Adult bees infected with the disease are seen with their wings open, shaky, and drooping. The body hairs of the bees fall out and the abdomen takes a dark and shiny color [66]. The abdomens of paralyzed bees with molting hairs appear smaller than they normally are. Due to chronic bee paralysis, they are unable to empty their intestines, leading to bloated abdomens and full honey sacs. Dysentery can occur as a result of these conditions. Bees infected with the disease do not fly and crawl around the hive in groups. When the hive is smoked, it becomes unable to move. Paralyzed bees are not taken into the hive by healthy bees after the flight. Diseased bees start to tremble after 1-2 days of being outside. The diseased bees that are not allowed inside gather in front of the hive and then die. In adult bees, deaths begin after a few days after the symptoms appear. When the severity of infection increases, sudden deaths can occur and colonies can be destroyed as a result. Heat and drought increase the multiplicity of the disease. It is not known how the disease is transmitted and it is estimated that it is transmitted during food transfer, as in other bee diseases. Chronic bee paralysis is usually caused by pesticides used by beekeepers. Another reason for the disease to be effective is the presence of old queens in the colony. The colony can be saved by queen replacement. No medicine can be used to detect the disease yet.

3.5. Deformed wing virus (DWV)

The deformed wing virus was first found in Japan [67]. It is noticeable that the wings of diseased baby bees are weak or deformed when emerging from the comb cells. Egyptian bee virus has a slight structural similarity with the deformed wing virus. The wing development in the honeycomb of the infected bees is disrupted and the structure changes. In the disease; abdominal shrinkage, wrinkling of the wings, and a decrease in mass are observed [68],[69]. Death is observed in larval and pupal stages [70]. Adult bees die without showing symptoms when infected [71]. Deformed wing virus is lethal on its own, when combined with varroa, it causes large colony losses [72],[73].

3.6. Slow paralysis virus

The slow paralysis virus was first discovered during laboratory research. This virus causes the death of the adult bee within 12 hours. Before the bee dies, the first pair of its legs are paralyzed [74]. When combined with varroa mites in the colony, it causes death in adult bees and larvae. As long as the varroa mite is present, it will feed on the bees and the slow paralysis virus will continue to reproduce.

3.7. Black queen bee virus

The black queen cell virus belongs to the *Dicistroviridae* family. This virus was identified as a result of encountering

dead queen larvae or pupae. The color of pupae and larvae that die as a result of the black queen virus is between dark brown and black. The infected larvae turn yellow and have hardened bag-like skin. Like the sacbrood virus, it multiplies very much when the pupa is infected. The virus does not reproduce when the bees eat the infected larvae. It was discovered in the USA by Clark in 1978. No known symptoms are known. When the black queen virus is combined with *nosema apis*, its reproduction is only accelerated. When this virus combines with the Y virus, the survival time of bees decreases. When combined with *nosema apis* and parasites in overwintering hives, the hive terminates rapidly. In a similar situation, when this virus combines with the filamentous virus, its severity decreases. Black queen virus is widespread in the world on its own and in combination.

3.8. Bee X virus

Bee X virus, which has links with the Y virus, is very difficult to distinguish from the Y virus. It shows high physical and chemical similarity. Bee X virus shows a disease effect as a result of the adult bee digesting infected products. This effect disrupts intestinal functions as in the Y virus. It does not interact with *Nosema apis* and this behavior distinguishes it from the Y virus. Bee X virus is most effective in the winter months. Bee X virus and *Malpighamoeba mellificae* parasite are found in bees that die in winter. Bee X virus is transmitted through bee feces and infected bees live less. If Bee X Virus and *Malpighamoeba mellificae* are found together, the disease reaches its most effective range. It is the most deadly range. When the disease reaches this level, it is extremely dangerous for the hives. Therefore, since new worker bees cannot be raised instead of young worker bees, life in the hive comes to an end. Bee X virus affects honey bees in Australia, Canada, Europe, Argentina, and Iran.

3.9. Cloudy wing virus

Cloudy wing virus belongs to the *Dicistroviridae* family. It is a common disease in bees. It causes the wings to appear opaque. Its particles are very small and can easily pass from hive to hive through the air. Cloudy wing virus is transmitted to the pupa by varroa. The disease can only be understood by serological tests. With the immunodiffusion technique, viruses that multiply in the head and thorax of the bee are determined. When the proportion of sick bees in the hive increases, bee vitality begins to disappear and the hive dies. It does not need certain seasons to reproduce and spread.

3.10. Apis iridescent virus

The disease is linked to the clustering disease of *Apis cerana* bees in India. Bees infected with the Apicidescent Virus do not want to work in summer, they cluster in the hive. Bees leave the hive and crawl on the soil. If more bees come out, the symptoms of the disease may decrease. This virus multiplies in the digestive system, hypofrangel glands, and ovaries. ApisIridescent Virus affects *Apis cerana* and *Apis mellifera*.

3.11. Israeli acute paralysis virus (IAPV)

Discovered in 2002 in Israel, the Israeli acute paralysis bee virus belongs to the *Dicistroviridae* family. It has a high genetic diversity. The most prominent feature of the disease is that the bees leave the hive before winter. There are too many dead bees in the hive. If there is not plenty of nectar around the bees, the virus reproduces too much. The bees stop feeding and brood care. This allows the bees to become early foragers. Healthy and young queens should be transplanted to colonies with the disease [75].

4. Protozoal diseases

4.1. *Nosema* disease (Nosemosis)

Nosema disease is a disease seen in most parts of the world and is caused by spore-bearing protozoa called *Nosema apis* [76]. The causative agent is an internal parasite [77]. *Nosema apis* spores are oval-shaped [78]. This disease is seen in adult honey bees [79]. The spores, which enter the digestive system through food and mouth, develop in the epithelial cells in the stomach and intestine of adult bees [80]. As a result of the cold and rainy weather, bees stay in the hive for a long time. Therefore, they cannot defecate outside the hive. Defecation inside the hive causes the parasite to increase. Infected bees in the colony transmit the disease to healthy bees through feces. Spores multiply rapidly in the digestive tract of the bees. Symptoms of the disease include separation of the wings, swelling of the abdomen, loss of sting reflexes, and the ability to fly [81]. They try to walk with the help of the grass in front of the hive. This disease, which starts to show its effect in the stomach, turns the stomach, which is straw-colored when healthy, into a dirty and white color. The feces of the bees are watery and sticky and yellow-white in color. In infected colonies; there are white and orange bee feces on combs, frames, and flight boards. The effects of the disease increase if the colony is queenless, malnutrition, insufficient pollen or nectar sources, or varroa disease in the colony. The use of infected combs, hive confusion, transfer of sick bees to other hives, excessive humidity in the hive, insufficient food, feeding with poor quality food, and windy and cold weather are effective in the spread of the disease. This disease reduces productivity by causing disorders in the digestive system of bees (figure 3). It causes the number of colonies to decrease. It has been determined that this disease agent can survive for at least 1 year in contaminated bee feces, 11 months in honey, 7 years under suitable conditions in the laboratory, and is resistant to cold, freezing, and microwaves. The disease is more common in humid environments and in spring months. The disease recovers when climatic conditions improve. Infected colonies have low honey production and a shortened life span [82]. For the diagnosis of the disease, microscopic and macroscopic examination of the stomach of the sick bee is required. The use of antibiotics is allowed in the treatment of the disease. Drugs with the active ingredient Fumagillin are given to the bees together with syrup. It is important to use the drug at the specified doses and time to avoid residue in the honey. After treatment of sick colonies with the drug, they should be transferred to a different hive that has been disinfected. Hives should be given food such as pollen cake and sugar syrup in the spring season. Strong colonies should be established. Apiaries should be established in airy and moisture-free places.



Fig 3. Examination of the digestive tract of adult honey bees, (A) control group, (B) nosema-infected group (Reprinted with permission from [83], Copyright MDPI).

4.2. Ordinary diarrhea (Dysentery)

It is when adult bees have diarrhea due to digestive disorders. It is not contagious and does not contain germs. The cause of the disease is improper feeding. It is more common in the spring season when bee activities begin. Since it is a seasonal disease, it goes away on its own as the season progresses. The disease passes with the increase in the amount of pollen and nectar in the environment. The disease can occur due to different reasons. Bees staying in the hive for a long time on rainy and windy days and therefore not being able to go on cleaning flights, feeding with dirty or rancid syrups, fermented foods, feeding bees with expired, spoiled syrups, damp cold, or humid environment, feeding with raw sugar, feeding bees with pollen due to the lack of sufficient honey in the hive in winter can be counted as the main causes of the disease [84]. In addition, the disease is also triggered by increased stress factors of the bees due to the constant disturbance of the wintering bees and disruption of their order: Bees are lethargic, they produce dark yellow, sticky, watery, and foul-smelling feces, and when the feces come into contact with the air, they dry up and lose their odor within 3-4 days. The source of the watery feces that accumulate in the intestines of bees is water. Normally, the digestive waste that accumulates in the intestines is excreted from the body during the spring cleaning flight away from the hive. The bee's intestine has a high ability to expand. Therefore, in winter, bees can carry and store feces in their intestines for a long time. Digestive residues accumulate in the bee's intestine up to a certain capacity. The accumulated feces remain in the honeycombs and contaminate the interior of the hive because of the lack of cleaning flights in rainy and windy weather. The water content in the feces of bees with dysentery is higher than in healthy bees. Bees infected with the disease cannot go out if the weather is cold. In this case, sick bees that stay in the hive for a long time defecate inside the hive. In this case, the inside of the hive becomes damp, moldy, and smelly and bee deaths begin to be seen. To treat the disease, a drop of formic acid and a small amount of alcohol can be added to the syrup and given to sick bees. If the disease is mild, it is not very harmful to the colony. But if it progresses together with nosema disease, its spread accelerates. If the disease progresses rapidly, mass deaths begin in the hive. Bees should be fed with refined granulated sugar. Fall maintenance and feeding should be done on time and should not be delayed. Fermented sour honey should not be given to the bee. Ventilation in the hive should be sufficient. To remove excess water, bees should be given time before the weather gets too cold. In addition, care should be taken in the feeding and care of the bees.

4.3. Amoeba disease

Malpighamoeba mellificia protozoan is the cause [85],[86]. It is found in the Malpighi tubes of honey bees infected with *Nosema apis*. It belongs to the class *Rhizopoda*. It is a very common protozoan. It is found on all continents where the honey bee is found. *Malpighamoeba mellifera* infects adult worker bees. Drones and queen bees are rarely infected. *M. Mellifera* in feces infects when eaten by bees. It is an extracellular parasite. It settles in Malpighi tubes and metamorphoses into cysts [87]. There is no typical symptom. It destroys the epithelial cells of the infected Malpighi tubes. In hives infected by *Malpighamoeba mellifera*, life is terminated. It is much more effective with *Nosema apis*. It can be diagnosed by microscopic examination of cysts taken from Malpighi tubes. Malpighi tubes are taken from the middle and posterior intestines. The size of the cysts is 5-8mm wide. The fight against the disease starts with the cleaning of beekeeping equipment. There are no drugs that can control it yet. When feces are found in the combs, they should be cleaned with acetic acid. In the early infected period, bees can be moved to clean hives.

4.4. Gregarines

Leidyana species of the Gregarine class targets honeybees. It acts in the digestive system of honey bees. It attaches to the intestinal epithelium through its organelles. It survives in the bee and continues to reproduce, producing the spores necessary to infect other bees. It is not specific to honey bees, it can be seen in other bees. It is transmitted to bees through nectar and water. It was first found in Europe. It is more common in temperate regions. Gregarines affect bee cells and cause changes. Diseased bees do not cause much damage to the colony. The disease can be managed by fumagillin application.

5. Mushrooms

5.1. Lime disease

Lime disease is a disease caused by a fungus called *Ascosphaera apis* [88]. The spores of the causative agent cause disease only in the larvae of bees and the reproduction of this disease is through sporulation. *Ascosphaera apis* heterotallic fungus is a type of fungus whose mycelia have male and female sexes. Mycelia of the two sexes must coexist for spores to form. Larvae acquire *Ascosphaera apis* through food. This agent develops in hives with insufficient ventilation due to the increase in the amount of CO₂ and the high level of humidity in the environment [89]. This disease is most common in spring and fall [90]. Strong colonies can overcome this disease in summer. These spores can live on the combs for years [91]. In addition, these spores, which can live inactive on the comb for years, become active again for reproduction if suitable conditions are provided. Drone larvae are usually found in the side combs of the hive and therefore remain at the lower level of incubation temperature. Since the optimum temperature for the development of this spore is 30°C, the first symptoms of the disease are seen in the male larvae on the edges of the combs. The diseased larvae mummify and dry inside the comb chamber. These larvae have a white, gray, or blackish color. In the later stages of the disease, white-colored mummified larvae are encountered because the disease spreads to the young in the middle parts of the comb. If the larvae are only infected with the female or the male, the infected bee is white like lime. However, if both the male and female are infected, the larvae are grayish-black.

The amount of moisture in the hive, environmental pollution, excessive humidity, stress factors such as starvation, use of adulterated beeswax, excessive use of syrup, inappropriate colony controls, feeding bees with artificial food, and swarming are effective in the formation of the disease. Improper cleaning of the hive by the bees is also one of the factors causing the disease [92]. There is an inverse relationship between bacteria and fungi. Antibiotics used to kill bacteria create favorable conditions for fungi to spread the disease. Spores of fungi are found on plants, in the soil, and between the body hairs of foraging bees, which are responsible for collecting pollen and honey. The fungi do not cause disease in adult bees but are transmitted to larvae by worker bees through food. Larvae are not infected for the first three days as they feed on royal jelly. When the bee larvae are 4-5 days old in open comb cells, infection is observed. As time progresses, the danger of the larvae becoming infected decreases. Deaths are most common during the mature larval and pupal stages. No chemicals or drugs are recommended for lime disease. In colonies with good cleaning behavior, larvae that tend to mummify or mummify are expelled from the hive by the worker bees in a short time. The diseased combs should be replaced with new combs in a short time so that the severity of the disease will be reduced [90]. Lines resistant to this disease should be used. Hives should be well-ventilated. The amount of moisture in the hive should remain at a certain level. Honeycombs from sick colonies should not be transferred to healthy colonies. Controls of colonies should not be carried out in the spring months when the weather is cool, and if it is necessary, the hive should not remain open for a long time. Perennial comb should not be used in colonies. The queens of sick colonies should be replaced with queens from healthy colonies. Work with strong colonies and young bees because weak colonies catch the disease faster.

5.2. Stone disease

It is also known as Stone Rot or Stonebrood. It is a rare disease [93]. The causative agent of this disease is a type of mold fungus called *Aspergillus flavus* [94]. This agent causes disease in adult and juvenile bees and causes death by infecting them. In the early stages of the disease, it is very difficult to diagnose the disease [95]. Besides *Aspergillus flavus*, *Aspergillus fumigatus*, and other *Aspergillus* species can also cause this disease [96]. These causal fungi are found in plants and soil. The disease affects the internal organs of the larvae and causes them to die. The larvae die in the closed honeycomb cells before entering the pupal stage. The larvae and pupae that carry the disease transform. *Aspergillus* first reproduces in the digestive tract. Then they gradually spread, covering the entire larva within a few days. After death, the larvae turn into mummies that are hard and hard to break like Stones [97]. The causative fungus

causes the body wall of the larva to burst, forming a false outer shell. Meanwhile, the outer surfaces of the larvae are covered with yellow fungal spores [98]. Bees that clean the honeycomb cells carry these spores and transmit the disease to other larvae, causing the disease to spread within the colony. Symptoms of the disease; bees look sluggish and restless. They have difficulty flying and are weak. The abdomen of the bees enlarges. The dead larvae and pupae dry up and become mummies. Honeycombs infected with this disease appear green. The spores of these fungi can live in honey and are therefore passed on to people who eat it. It is dangerous to eat honey from infected hives. In humans and other mammals, it causes infection of the upper respiratory tract. High humidity in the hive, disruption of the intestinal flora due to antibiotic use, inadequate ventilation in the hive, increase in the temperature of the hive are important factors in the emergence of the disease. In addition, an increase in the stress rate is also an effective factor in the emergence of the disease [99]. The disease can also be spread by carrying contaminated honeycombs to healthy colonies: There is no chemical treatment for this disease. It is necessary to disinfect the hives in which the disease is detected and burn the honeycombs. Honey infected with the disease is not suitable as food for humans and bees and should not be used. Hives and other tools used should be disinfected. Hive ventilation should be done well, high humidity should be prevented. The queen bee should be replaced.

It is found in the pollen in the honeycomb cells. Another name is pollen fungus. The disease is seen in early spring after the winter season. In hives where this disease is observed, lime disease is observed later. *Bettisia alvei* develops more easily at temperatures lower than 30°C. It multiplies in the pollen in the comb during winter and early spring. Pollen is damaged and wasted. Honeycombs are closed after they are filled with honey, but if the honeycombs are not completely filled with honey or if honey is removed from the honeycombs and closed, a very good environment is created for *Bettisia alvei* to multiply. When the disease takes effect, pollen becomes unusable. Summer heat is not effective for spores. It is more common in weak colonies during cold times. The disease turns pollen into hard and dry particles. The fungus in the honeycombs is gray and is found in pollen clusters. Bees expel these pollen clusters. *Bettisia alvei* is not a problem in healthy colonies. It does not harm the normal cycle of the bees. In warm weather, the problematic pollen is removed from the comb, and the disease is combated. To prevent the onset of *Bettisia alvei*, it is necessary to keep the hives dry and wrapped in winter.

5.4. Other mushrooms

Fungi are usually collected by foraging bees. Most fungi collected by foraging bees cannot survive in hives and are not effective for bees. Some fungi can reproduce after the life of the bees is over and cause mummification of soft tissues. Fungi can be harmful under favorable conditions, but they can be a problem for weak colonies if they are provided with favorable conditions of humidity, temperature, and low ventilation. *Mucor mucedo* and *Trichoderma lignorum* cause disease in adult bees and larvae. *Aspergillus niger* causes disease in queen larvae. *Rhizopus equinus*, seen in adult bees and larvae, is caused by contamination of flowers with horse manure. It is seen in cold countries. *Scopulariopsis brevicaulis*, infected larvae appear black or yellow and are found adhered to the honeycomb walls. The disease disappears when the temperature is effective. *Labyrinthula apis* and *Endomycopsis apis* cause larval death.

6. Honey bee pests

6.1. Yeasts

Yeasts are not considered a disease agent for honey bees. The preservation of bee bread provides growth factors and vitamins. *Candida utilis* is very useful for young bees. Some osmophilic yeasts are fermentation agents in honey and change the structure of honey. As a result, malnutrition is seen in adult bees and larvae and deaths occur.

6.2. Varroa

Varroosis disease is caused by the general pathogen called *Varroa jacobsoni oudemans* [118]. This disease causes deformation of the wings and legs of the bees, shortness of the legs, short abdomen, low productivity, and easier transmission of other diseases. The female varroa is oval in appearance, dark brown in color, and the mouth structure is stinging and sucking. The male ones are yellow or gray in color and round in appearance. It has a softer chitin structure than the female varroa. Male varroas are not seen on the bee when they die after mating with the female. The reproductive period of varroa in colonies starts with the brood period in spring [100]. With the end of the brood period in the fall, the reproduction of varroa also ends. Varroa reproduce and develop in closed brood cells in the colony. Adult female varroes enter the brood cells just before they close and start laying eggs two days later. In the first 24 hours, 6-legged larvae hatch from the eggs. The larva completes its development in 6-7 days if it is male and 8-10 days if it is female. Varroa feeds on the hemolymph of larvae, pupae, and adults, which is the equivalent of blood in invertebrates. As a result, pupae do not develop well and adults are weak and unable to fly (figure 4). The number of drones in the colony drops noticeably. The ability of drones to mate is also considerably reduced. The lifespan of the bees in the colony shortens. Worker bees are smaller than their normal size. This size reduction is more pronounced during the pupation period. Young bees emerging from the cells may have wingless, single, or short wings. In addition to these, abnormalities such as missing legs and a short abdomen are also seen.



Fig 4. (A) examination of drone larvae showing red lava with Varroa mites, (B) Varroa mites on the thorax of drones (Reprinted with permission from [101], Copyright MDPI).

The worker bees' brood care is weakened and therefore the queen's oviposition capacity is reduced. The wounds opened by varroa on the bees to meet their nutritional needs create a favorable environment for various diseases. Since they feed on hemolymph, the resistance of bees against diseases decreases. Since Varroa has many ways of spreading and adapts to colony biology quickly and easily, it becomes difficult to fight. Control methods are classified as physical, biological, and chemical. Physical and biological control is usually carried out in spring and summer, while chemical control is carried out in fall and winter. Varroes die between %74-98 at 46°C. For this reason, in specially prepared hives, the temperature is increased in a controlled manner and the varroas are expected to die and fall to the bottom board. Female varroas prefer drone cells to lay eggs in the spring period. In the spring period, honeycombs with drone cells are placed on the colonies and female varroa are collected in the drone cells. After these eyes close, they are removed from the hive and destroyed. In this way, the eggs laid by the female varroa and the drone pupae are

destroyed. Thanks to this method, it is possible to reduce the number of varroa in the colony. In addition, it continues to be effective because the varroa that multiply in the worker bee cells remain. It is the control of mites with chemicals used for the control of mites. Things to be considered in acaricide applications; Chemical substances that have not been laboratory tested or that have negative results for human or bee health should not be used, the desired drugs should be used as described and in dosage, and acaricide should not be used during the harvest period. Spraying should be done in spring and fall periods, spraying should be done on days when the air temperature is higher than 14°C, and in periods when the bees come from nectar collection, spraying should be done after removing the partitioned boards in the hive to facilitate the contact of the drugs with the bees [102].

6.3. Small hive beetle (*Aethina tumida*)

The small hive beetle, a very dangerous pest, was first found in the USA in 1998. Tens of thousands of colonies were destroyed. The small hive beetle, which spread very rapidly, became the most important problem of American beekeeping in those years. It is thought to have arrived with vegetables and fruits imported from Africa around 1995 [103]. This insect originated from South Africa and belongs to the family Nitidulidae of the order *Aethina tumida Coleoptera* [104], [105], [106]. African bees are not affected by the small hive beetle due to their cleaning properties. Since it is harmless in Africa, it was almost unrecognized until 1988. Physiologically, adults are about 5-7 mm long, 6-legged, reddish brown or black, and have 2 pairs of wings [107], [108]. Their bodies are protected from bee stings by a thick layer of chitin. Covered with very fine hairs, the small hive beetle is very difficult to catch by bees. The adult beetles can fly and move between hives. Flight distances have not been determined. They reproduce by laying eggs. Their eggs, similar to honey bees, are pearl white. They can lay eggs almost all the time except in harsh winters. Larvae are cream-colored, about 11mm long, and about 1.6mm in diameter. The larvae cannot spin cocoons but instead are surrounded by a thick layer of chitin. The thick layer of chitin protects the larvae from bees. They can reproduce continuously in the hive and even take over the hive and neutralize the hive. Larvae use honey and pollen as food [109], [110]. They eat the glaze on the honey and defecate on the honey. Sour, rotten orange-smelling honey flows out of the hive. The bees do not touch this contaminated honey, but if it covers the honeycomb, they move away from the honeycomb. It has been found that bees return only when the honeycomb is cleaned with water. When the adult reaches the size of an insect, it leaves the hive, goes under the soil and the pupation period begins [111]. The pupae are as big as the adult beetle and have an amber or blue color. The life of the small hive beetle begins when the female beetle lays eggs in the honeycomb in an irregular way. The larvae hatch from these eggs in about 2-3 days. Even a very small number of female beetles are effective because they can lay a very large number of eggs. After the larval period lasting 10-16 days, they leave the hive and enter the soil. 3-4 weeks pass after the pupation period begins and adult beetles are formed. The life span of adult beetles is approximately 6 months. Adult beetles lay eggs in their first week. They can produce up to 5 generations per year. There is no control mechanism against the small hive beetle yet. Small hive beetles die when kept at -12 degrees Celsius for 24 hours.

6.4. Bee louse

It is not actually a species of louse but a member of the order *Diptera* [112]. The insect that causes this disease is *Braula coeca* [113],[114]. This is a small, colorful insect. It is shiny and brownish-red in color. This insect has no wings and no eyes. This disease does not harm the bee directly, it is a food partner. Adult bee lice are similar to varroa [115]. They are distinguished from varroa by their three pairs of legs and the licking-sucking mouthparts. Bee lice also have a distinguishable head structure when viewed from the top. They live on worker and queen bees. They survive by clinging to the hairs on the chest of bees. It is very rarely seen on drones. Adult bee lice are found on the chest and mouth parts of bees and steal food from the bees' mouths. They do not live by sucking the blood of bees but by feeding on royal jelly, pollen, and honey [116]. Since royal jelly is their favorite food, they share the food of the queen bee and reduce its performance. The adult bee louse is 1.5 mm long and 1 mm wide. Male lice are smaller than female lice. Adult females of bee lice lay their eggs in the upper part of the comb cells. The egg is white in color and

oval in shape. Bee lice can lay their eggs anywhere in the colony. The larvae that hatch from the eggs pass through the honey glazes and consume the honey, which is their food source. It is more common in the fall and spring. It cannot reproduce in winter. Adult bee lice spend the winter on the bee. It is possible for 15-20 bee lice to live on a queen bee. This causes the queen to stop laying eggs. If more bee lice are found, the queen dies. The disease spreads due to swarming, predation, carrier bees getting confused in their own hives, and colony transplants. The bee louse causes the queen bees to become weak, their ability to lay eggs decreases, and the marketing value of honey decreases due to the deterioration of the honeycombs in the hive. Tobacco smoke is used in the control of bee lice. Cigarette tobacco burned in the bellows is given into the hive through the flight hole. Bee lice, which faint under the influence of smoke, fall to the bottom board and are collected and destroyed. Bromopropylate and formic acid are used as medicines.

6.5. Trachea parasite

It was first detected in England in 1921 by Rennie [117]. After being seen in England, it spread to various regions. It is a parasite called *Acarapis Woodi* and is also known as tracheal mite [118]. There are 3 *Acarapis* species in honey bees and these are *Acarapis woodi*, *Acarapis dorsalis*, and *Acarapis externus*. The harmful species among these species is *Acarapis woodi*. This disease is an internal parasite that settles on the respiratory system of worker bees and can lead the bees to death [119], [120]. It is sometimes seen in queens and drones. It has male and female sexes. It feeds and reproduces in the trachea, the respiratory tract. After the female and male mate, the male tracheal mite dies. The female tracheal mite enters through the first thorax of the adult bee and settles in the trachea. Within 1-2 days she can lay 6-10 eggs in the trachea. The eggs complete their development within 14 days. The larvae hatch from the eggs, pierce the wall of the trachea with their mouthparts, and use the bees' blood as food. This parasite first enters the trachea of 1-4 day-old young bees and sucks their blood. When blood leaks from the trachea, the young bee's trachea becomes blocked or damaged. The bee, deprived of oxygen for these reasons, dies from respiratory failure. The disease has some symptoms. The most important symptom in bees infected with the tracheal mite is the inability of sick bees to fly [121]. They have asymmetrical wing positions. The wings appear drooping. The abdomen appears swollen. The trachea of a healthy bee appears pale, transparent, spotless and clear. The trachea of sick larvae, on the other hand, has crusting, brown spots and a black color depending on the number of mites. The lifespan of an adult tracheal mite is around 30-40 days. In dead bees, the tracheal mite can live for 1-2 days. For their development, 34°C is the optimal temperature. It develops the fastest in winter when it is in the hive. By the end of winter, it has contaminated the bee's trachea with its feces and eggs. When the bee takes its first flight, it moves a certain distance away from the hive and becomes unable to breathe air due to the clogged and damaged trachea, and death occurs. Domestic Anatolian breed bees do not get this disease because they are resistant to the parasite that causes the disease. For the diagnosis of the disease, diseased bees should be examined in the laboratory and a definite conclusion should be reached afterwards. Treatment methods used in varroa disease can be used in its treatment.

6.6. Lepidoptera

6.6.1. Large wax moth

Galleria mellonellae, known by different names such as bee moth, honeycomb moth, large wax moth, etc., cause damage to honey and combs [122],[123]. It is usually found in weak bee colonies without queen bees and weakened bee colonies [124]. In weak colonies, there is a decrease in brood, honey, and pollen production due to the residues and nets of *Galleria mellonella*. When the temperature increases, hives begin to die out. The development of the moth is accelerated by lack of food, absence of queen bees, varroa, and pesticide poisoning in colonies. Large wax moth is found all over the world [125]. Its harmfulness disappears at high altitudes. Its presence decreases when the altitude is above 1000 meters, and it cannot survive when the altitude exceeds 1500 meters. In Turkey, it is found in regions with warm and mild climates.

6.6.2. Small wax moth (*Achroia Grisella*)

It is also known for its *Achroia grisella* [126], [127], [128]. It is more harmless than the large wax moth. It is effective in weak colonies [129]. Its physical characteristics are light yellow on the head, silver gray on the body and male moths are 10mm in size and female moths are 13mm in size. Females usually live for 7 days and produce 250-300 eggs during their lifetime. The developmental stage is similar to that of the large wax moth and it makes tunnels made of silk. The small wax moth eats combs with young, honey, pollen, bee brood, and beeswax [130]. It does not cause as much damage as the large wax moth. In hives with the small wax moth, the comb cells are not closed even though there are bee larvae in the comb cells. The reason for this is that the moth has eaten the honeycomb wall and made a hole, and the worker bees remove the entire honeycomb wall instead of repairing the hole. The honeycombs that remain open contain moth feces, and the bee larvae that can grow under these conditions show developmental disorders.

6.6.3. Bumble bee wax moth

Aphomia sociella, the Bumble Bee wax moth, is found in Europe and Asia [131], [132]. It is found in the nests of bumblebees and rarely in honey bee colonies. Adults are slightly smaller than the large wax moth. Mature larvae are yellow and 22-30 mm in size. Larvae eat bee eggs, larvae, pupae, pollen, and honey [133].

6.6.4. Deadhead moth

Acherontia atropos feeds by eating honey or nectar in bee colonies. The deadhead moth attacks bee colonies in the evening and stays inside. Sometimes dead moths are found inside the hive.

6.7. Ants

Ants, which live in almost every region of the world, can be a problem for honey bees. The carpenter ant gnaws the wooden parts of the hives. When other ants find the opportunity, they enter the hive and eat the honey, pollen and baby bees, and when their numbers are dominant, they also eat the adult bees.

6.8. Wasps

Wasps attack colonies in beekeeping regions and cause serious economic consequences. It is a problem in East Asia, Southeast Asia, and the island of Japan. *Apis cerana*, *Apis florea*, and *Apis mellifera* colonies are more affected by wasps.

6.9. Birds

Birds prefer small or large insects for feeding and bees are also in the food chain. Birds are known to hunt bees during migrations, during accommodation, and in the areas where they live.

6.10. Moxibustion beetle (*Meloidea*)

It belongs to the *Coleoptera* order and *Meloidae* family. The most important and harmful species known in our country are *Meloe variegates*, *M. proscarabeus*, *M. vilaceus*, and *M. cavensis*. They act on bees in two different ways. Firstly, they eat eggs and larvae. Secondly, they parasitize mature bees. 1st instar larvae feed on the hemolymph of bees; 2nd

instar larvae consume honey, pollen, bee eggs, and bee larvae as food. The adults of this species do not harm honey bees. Moxibustion beetles are found in various regions such as the Aegean, Mediterranean, Central Anatolia, and Black Sea regions. *Meloe violaceus* Marsham species are effective in the Erzurum region in our country. The length of adult bees is around 22-30 mm. Male bees are smaller and female bees are larger. Moxibustion beetles consume plant leaves and stems as food during their adult stages. However, there is not yet a successful intervention method with the moxibustion beetle [134].

7. Nanotechnology and bee diseases

Nanotechnology science is used in many fields today [135]- [167]. Nanomaterials are reported in the literature to be able to have antibacterial, antifungal, and antiviral properties [168], [169], [170], [171]. The use of nanomaterials smaller than 100 nanometers (nm) for specific purposes has been applied in agricultural, industrial, and medical fields [172]. Therefore, nanotechnology has many potential applications in beekeeping as well as in various fields. Nanotechnology is attracting attention as an advanced tool in the diagnosis and treatment of bee diseases. Targeted and pest-directed therapies formulated with nanoparticles are available. With antimicrobial properties such as silver nanoparticles, they can protect bees against pathogens in a highly functional way [173]. Thus, the positive impact on bee health and lifespan is a remarkable research topic [174]. These technologies have the potential to offer sustainable solutions in beekeeping.

8. Conclusion

Even though beekeeping is a very important sector dating back to the past, there are many inexperienced producers engaged in beekeeping. In addition, bees can be affected by many biotic factors such as ecological and geographical abiotic factors, natural disease agents, and predators. Apart from these reasons that affect the damage of bees, parasites, bacterial, viral, and fungal diseases cause many negativities in the sector. Among these environmental and biological effects, the factors that cause natural diseases in bee colonies cause large colony losses. Therefore, the most important point in bee diseases is to protect the hive with the right information, equipment, and methods. Diseased hives should definitely be separated and transported, weak hives should be combined, and procedures that will strengthen the hive, such as the reinforcement of frames with brood, should be followed meticulously. All equipment used in beekeeping should always be kept clean and go through sterilization stages. Hives should be strengthened and regularly maintained, and bees should be fed with vitamin syrups and cake when necessary. Thus, healthy growth and body resistance of the bees should be increased and the diseases should be minimized. Methods of controlling other predators, insects, and parasites should be sought. Various chemicals, antibiotics, and natural methods are at the top of these control methods. Potassium hypochlorite and 2% formaldehyde solution are the most commonly used in honeycomb disinfection. However, honey and combs in the hive should not be given to the bees after chemical use. Potassium hypochlorite is frequently used for sterilization of metal beekeeping equipment. Other chemicals such as benzalkonium chloride, hydrogen peroxide, and chloramine are used to prevent and treat bee diseases.

Acknowledgments

The authors dedicated this publication to the 100th anniversary of the Republic of Türkiye. As scientists raised by Türkiye, they are proud to be citizens of this country.

Author Contribution

Ebru Halvacı, Mert Gül, Hatice Kars, Teslime Kozak; Writing - original draft, Editing, Formal analysis. Damla İkbali, Alper Özgünlü; Visualisation, Formal analysis Fatih Şen; Writing - review and editing, Verification, Research, Funding

References

- [1] S. Nekoei, M. Rezvan, F. Khamesipour, C. Mayack, M. B. Molento, and P. D. Revainera, "A Systematic Review of Honey Bee Infections and Available Treatment Options," *Vet. Med. Sci.*, vol. 9, no. 4, pp. 1848–1860, Jul. 2023, doi: 10.1002/vms3.1194.
- [2] B. Olas, "Bee Products as Interesting Natural Agents for the Prevention and Treatment of Common Cardiovascular Diseases," *Nutrients*, vol. 14, no. 11, p. 2267, May 2022, doi: 10.3390/nu14112267.
- [3] E. Amiri, M. Strand, O. Rueppell, and D. Tarpy, "Queen Quality and the Impact of Honey Bee Diseases on Queen Health: Potential for Interactions between Two Major Threats to Colony Health," *Insects*, vol. 8, no. 2, p. 48, May 2017, doi: 10.3390/insects8020048.
- [4] H. Kim, S.-Y. Park, and G. Lee, "Potential Therapeutic Applications of Bee Venom on Skin Disease and Its Mechanisms: A Literature Review," *Toxins (Basel)*, vol. 11, no. 7, p. 374, Jun. 2019, doi: 10.3390/toxins11070374.
- [5] J. Ebeling, A. Fünfhaus, and S. Gisder, "Special Issue: Honey Bee Pathogens and Parasites," *Vet. Sci.*, vol. 9, no. 10, p. 515, Sep. 2022, doi: 10.3390/vetsci9100515.
- [6] H. Tutun, N. Koç, and A. Kart, "Plant Essential Oils Used Against Some Bee Diseases," *Turkish J. Agric. - Food Sci. Technol.*, vol. 6, no. 1, pp. 34–45, Feb. 2018, doi: 10.24925/turjaf.v6i1.34-45.1502.
- [7] P. J. Marín-García, Y. Peyre, A. E. Ahuir-Baraja, M. M. Garijo, and L. Llobat, "The Role of Nosema Ceranae (Microsporidia: Nosematidae) in Honey Bee Colony Losses and Current Insights on Treatment," *Vet. Sci.*, vol. 9, no. 3, p. 130, Mar. 2022, doi: 10.3390/vetsci9030130.
- [8] M. Goblirsch, "Nosema Ceranae Disease of the Honey Bee (*Apis Mellifera*)," *Apidologie*, vol. 49, no. 1, pp. 131–150, Feb. 2018, doi: 10.1007/s13592-017-0535-1.
- [9] A. G. Dolezal and A. L. Toth, "Feedbacks Between Nutrition and Disease in Honey Bee Health," *Curr. Opin. Insect Sci.*, vol. 26, pp. 114–119, Apr. 2018, doi: 10.1016/j.cois.2018.02.006.
- [10] M. Sarwar, "Fungal Diseases of Honey Bees (Hymenoptera : Apidae) That Induce Considerable Losses to Colonies and Protocol for Treatment," *Int. J. Zool. Stud.*, vol. 1, no. 1, pp. 8–13, 2016.
- [11] K. Kuzyšinová, D. Mudroňová, J. Toporčák, L. Molnár, and P. Javorský, "The Use of Probiotics, Essential Oils and Fatty Acids in the Control of American Foulbrood and Other Bee Diseases," *J. Apic. Res.*, vol. 55, no. 5, pp. 386–395, Dec. 2016, doi: 10.1080/00218839.2016.1252067.
- [12] E. Halvacı, T. Kozak, M. Gül, H. Kars, and F. Şen, "Bee Anatomy: A Comprehensive Overview of Bee Morphology and Physiology," / *J. Sci. Reports-B*, vol. 8, pp. 1–19, 2023.
- [13] J. Ebeling *et al.*, "A Comparison of Different Matrices for the Laboratory Diagnosis of the Epizootic American Foulbrood of Honey Bees," *Vet. Sci.*, vol. 10, no. 2, p. 103, Feb. 2023, doi: 10.3390/vetsci10020103.
- [14] M. Djukic *et al.*, "Comparative Genomics and Description of Putative Virulence Factors of *Melissococcus plutonius*, the Causative Agent of European Foulbrood Disease in Honey Bees," *Genes (Basel)*, vol. 9, no. 8, p. 419, Aug. 2018, doi: 10.3390/genes9080419.
- [15] A. Gregorc and B. Sampson, "Diagnosis of Varroa Mite (*Varroa destructor*) and Sustainable Control in Honey Bee (*Apis mellifera*) Colonies," *Diversity*, vol. 11, no. 12, p. 243, Dec. 2019, doi: 10.3390/d11120243.
- [16] K. S. Traynor *et al.*, "Varroa Destructor: A Complex Parasite, Crippling Honey Bees Worldwide," *Trends Parasitol.*, vol. 36, no. 7, pp. 592–606, Jul. 2020, doi: 10.1016/j.pt.2020.04.004.
- [17] J. M. Flores *et al.*, "Impact of Varroa Destructor and Associated Pathologies On the Colony Collapse Disorder Affecting Honey Bees," *Res. Vet. Sci.*, vol. 135, pp. 85–95, Mar. 2021, doi: 10.1016/j.rvsc.2021.01.001.
- [18] J. Wegener *et al.*, "Pathogenesis of Varroosis at the Level of the honey bee (*Apis Mellifera*) Colony," *J. Insect Physiol.*, vol. 91–92, pp. 1–9, Aug. 2016, doi: 10.1016/j.jinsphys.2016.06.004.
- [19] F. Masood *et al.*, "Evaluating Approved and Alternative Treatments Against an Oxytetracycline-Resistant Bacterium Responsible for European Foulbrood Disease in Honey Bees," *Sci. Rep.*, vol. 12, no. 1, p. 5906, Apr. 2022, doi: 10.1038/s41598-022-09796-4.
- [20] A. J. Burnham, "Scientific Advances in Controlling Nosema Ceranae (Microsporidia) Infections in Honey Bees (*Apis mellifera*)," *Front. Vet. Sci.*, vol. 6, Mar. 2019, doi: 10.3389/fvets.2019.00079.
- [21] J. Belsky, D. J. Biddinger, N. Seiter, and N. K. Joshi, "Various Routes of Formulated Insecticide Mixture Whole-Body Acute Contact Toxicity to Honey Bees (*Apis Mellifera*)," *Environ. Challenges*, vol. 6, p. 100408, Jan. 2022, doi: 10.1016/j.envc.2021.100408.
- [22] E. Halvacı, T. Kozak, M. Gül, H. Kars, R. Bayat, and F. Şen, "Journey to the Sweet World of Beekeeping : Historical Development , Honey Harvesting and Overview of Bee Products," pp. 39–58, 2024.
- [23] E. Genersch, J. D. Evans, and I. Fries, "Honey bee disease overview," *J. Invertebr. Pathol.*, vol. 103, pp. S2–S4, Jan. 2010, doi: 10.1016/j.jip.2009.07.015.
- [24] J. D. Hitchcock, J. O. Moffett, J. J. Lockett, and J. R. Elliott, "Tylosin for Control of American Foulbrood Disease in Honey Bees134," *J. Econ. Entomol.*, vol. 63, no. 1, pp. 204–207, 1970, doi: 10.1093/jee/63.1.204.

- [25] M. Ye, X. Li, F. Yang, and B. Zhou, "Beneficial bacteria as biocontrol agents for American foulbrood disease in honey bees (*Apis mellifera*)," *J. Insect Sci.*, vol. 23, no. 2, pp. 1–5, 2023, doi: 10.1093/jisesa/iead013.
- [26] J. G. Stephan, J. R. De Miranda, and E. Forsgren, "American foulbrood in a honeybee colony: Spore-symptom relationship and feedbacks," *BMC Ecol.*, vol. 20, no. 1, pp. 1–14, 2020, doi: 10.1186/s12898-020-00283-w.
- [27] M. Spivak and G. S. Reuter, "Resistance to American foulbrood disease by honey bee colonies *Apis mellifera* bred for hygienic behavior," *Apidologie*, vol. 32, pp. 555–565, 2001.
- [28] E. Borum, "Arıların Yavru Çürüklüğü İnfeksiyonlarında Doğru Teşhis, Mücadele ve Korunma Yöntemleri," vol. 14, no. May, pp. 44–55, 2014.
- [29] Ş. Ö. Uygur and A. O. Girişgin, "Bal Arısı Hastalık ve Zararlıları," *Uludağ Arıcılık Derg.*, vol. 8, no. 4, pp. 130–142, 2008.
- [30] M. López-Urbe and R. Underwood, "Honey Bee Diseases: American Foulbrood," *PenneState Ext.*, no. 1, pp. 1–6, 2022.
- [31] A. T. Truong *et al.*, "Probiotic candidates for controlling *Paenibacillus* larvae, a causative agent of American foulbrood disease in honey bee," *BMC Microbiol.*, vol. 23, no. 1, pp. 1–11, 2023, doi: 10.1186/s12866-023-02902-0.
- [32] M. Moharrami, N. Mojani, M. Bagheri, and S. Toutiaee, "Role of Honey Bee Gut Microbiota in the Control of American Foulbrood and European Foulbrood Diseases," *Arch. Razi Inst.*, vol. 77, no. 4, pp. 1331–1339, 2022, doi: 10.22092/ARI.2022.358073.2146.
- [33] E. Forsgren and A. T. Laugen, "Prognostic value of using bee and hive debris samples for the detection of American foulbrood disease in honey bee colonies," *Apidologie*, vol. 45, no. 1, pp. 10–20, 2014, doi: 10.1007/s13592-013-0225-6.
- [34] R. M. Alonso-Salces *et al.*, "Natural strategies for the control of *Paenibacillus* larvae, the causative agent of American foulbrood in honey bees: a review," *Apidologie*, vol. 48, no. 3, pp. 387–400, 2017, doi: 10.1007/s13592-016-0483-1.
- [35] M. Alburavi, S. K. Abban, J. D. Evans, and Y. P. Chen, "Occurrence and distribution of two bacterial brood diseases (American and European foulbrood) in US honey bee colonies and resistance to antibiotics from 2015 to 2022," *J. Apic. Res.*, vol. 0, no. 0, pp. 1–10, 2024, doi: 10.1080/00218839.2024.2329854.
- [36] M. F. Feldlaufer, J. S. Pettis, J. P. Kochansky, and G. Stiles, "Lincomycin hydrochloride for the control of American foulbrood disease of honey bees," *Apidologie*, vol. 32, no. 6, pp. 547–554, 2001, doi: 10.1051/apido:2001100.
- [37] H. Hansen and C. J. Brødsgaard, "American foulbrood: A review of its biology, diagnosis and control," *Bee World*, vol. 80, no. 1, pp. 5–23, 1999, doi: 10.1080/0005772X.1999.11099415.
- [38] D. C. De Graaf *et al.*, "Diagnosis of American foulbrood in honey bees: A synthesis and proposed analytical protocols," *Lett. Appl. Microbiol.*, vol. 43, no. 6, pp. 583–590, 2006, doi: 10.1111/j.1472-765X.2006.02057.x.
- [39] B. Bık, J. Szkoła, J. Wilk, P. Artiemi Jew, and J. Wilde, "In-Field Detection of American Foulbrood (AFB) by Electric Nose Using Classical Classification Techniques and Sequential Neural Networks," *Sensors*, vol. 22, no. 3, p. 1148, Feb. 2022, doi: 10.3390/s22031148.
- [40] I. Abdullah, S. R. Gary, and S. Marla, "Field trial of honey bee colonies bred for mechanisms of resistance against *Varroa destructor*," *Apidologie*, vol. 38, pp. 67–76, 2007, doi: 10.1051/apido.
- [41] A. S. Floyd, B. M. Mott, P. Maes, D. C. Copeland, Q. S. McFrederick, and K. E. Anderson, "Microbial ecology of european foul brood disease in the honey bee (*Apis mellifera*): Towards a microbiome understanding of disease susceptibility," *Insects*, vol. 11, no. 9, pp. 1–16, 2020, doi: 10.3390/insects11090555.
- [42] E. Forsgren, G. E. Budge, J. D. Charrière, and M. A. Z. Hornitzky, "Standard methods for European foulbrood research," *J. Apic. Res.*, vol. 52, no. 1, 2013, doi: 10.3896/IBRA.1.52.1.12.
- [43] P. R. S. Rajamanoharan and S. Vivekanandarajah Sathasivampillai, "The role of honey in pediatric treatments in Sri Lankan Siddha medicine," *Uludağ Arıcılık Derg.*, vol. 20, no. 2, pp. 83–90, 2021, doi: 10.31467/uluaricilik.781259.
- [44] E. Forsgren, "European foulbrood in honey bees," *J. Invertebr. Pathol.*, vol. 103, no. SUPPL. 1, pp. S5–S9, 2010, doi: 10.1016/j.jip.2009.06.016.
- [45] O. Lewkowski and S. Erler, "Virulence of *Melissococcus plutonius* and secondary invaders associated with European foulbrood disease of the honey bee," *Microbiologyopen*, vol. 8, no. 3, pp. 1–9, 2019, doi: 10.1002/mbo3.649.
- [46] M. T. Student *et al.*, "Nevşehir İli Merkez Karşıdağ Mevkii *Apis mellifera* (Insecta: Hymenoptera: Apidae) Taksonu Biyolojisi ve Morfolojisi Üzerine Araştırma," *Front. Neurosci.*, vol. 14, no. 1, pp. 1–13, 2021.
- [47] A. Wicaksana and T. Rachman, "Arı Hastalıkları," *Angew. Chemie Int. Ed.* 6(11), 951–952., vol. 3, no. 1, pp. 10–27, 2018.
- [48] K. Papadopoulou-Karabela, N. Iliadis, V. Liakos, and E. Bourdzy-Hatzopoulou, "Experimental infection of honeybees by *Pseudomonas aeruginosa*," *Apidologie*, vol. 23, no. 5, pp. 393–397, 1992, doi: 10.1051/apido:19920501.
- [49] M. A. Kırpık and C. Kadirhan, "Bacterial Diseases of Honey Bee (*Apis mellifera*)," *Sci. Technique 21st Century*, vol. 2, no. 3, pp. 1–18, 2015.
- [50] Ö. Ertürk and B. Taş, "Bacterial Flora Analysis in Mud Nests of Alien Invasive Wasps (*Sceliphron curvatum* Smith, 1870)," *Ordu Üniversitesi Bilim ve Teknol. Derg.*, vol. 13, no. 1, pp. 37–43, 2023, doi: 10.54370/ordubtd.1274632.
- [51] H. Q. Zheng and Y. P. Chen, "Detection of *Spiroplasma melliferum* in honey bee colonies in the US," *J. Invertebr. Pathol.*, vol. 119, pp. 47–49, 2014, doi: 10.1016/j.jip.2014.03.006.
- [52] M. Sarwar, "Challenges due to bacterial infections of the honey bees and contributions to manage pest problems," *Int. J. Entomol. Res. Issue*, vol. 1, no. 1, pp. 2455–4758, 2016.
- [53] P. E. Lee and B. Furgala, "Electron microscopy of sacbrood virus in situ," *Virology*, vol. 25, no. 3, pp. 387–392, 1965, doi: 10.1016/0042-6822(65)90059-0.
- [54] I. Balkaya, H. Gülbaz, H. Avcioğlu, and E. Güven, "Türkiye’de Görülen Bal Arısı (*Apis mellifera*) Hastalıkları," *Atatürk Univ. Vet. Bilim. Derg.*, vol. 11, no. 3, pp. 339–347, 2016, doi: 10.17094/ataunivbd.282993.
- [55] M. Mingxiao, L. Ming, C. Jian, Y. Song, W. Shude, and L. Pengfei, "Molecular and biological characterization of chinese sacbrood virus

- LN isolate," *Comp. Funct. Genomics*, vol. 2011, 2011, doi: 10.1155/2011/409386.
- [56] P. Tuncer and K. Yeşilbağ, "Bal Arılarının Viral Hastalıkları," *Arı Bilimi / Bee Sci.*, vol. 9, no. November, pp. 149–161, 2009.
- [57] P. Blanchard *et al.*, "Development and validation of a real-time two-step RT-qPCR TaqMan® assay for quantitation of Sacbrood virus (SBV) and its application to a field survey of symptomatic honey bee colonies," *J. Virol. Methods*, vol. 197, pp. 7–13, 2014, doi: 10.1016/j.jviromet.2013.09.012.
- [58] L. Bailey, "The multiplication and spread of sacbrood virus of bees," *Ann. Appl. Biol.*, vol. 63, no. 3, pp. 483–491, 1969, doi: 10.1111/j.1744-7348.1969.tb02844.x.
- [59] W. F. Huang, S. Mehmood, S. Huang, Y. W. Chen, C. Y. Ko, and S. Su, "Phylogenetic analysis and survey of Apis cerana strain of Sacbrood virus (AcSBV) in Taiwan suggests a recent introduction," *J. Invertebr. Pathol.*, vol. 146, pp. 36–40, 2017, doi: 10.1016/j.jip.2017.04.001.
- [60] R. C. Ghosh, B. V. Ball, M. M. Willcocks, and M. J. Carter, "The nucleotide sequence of sacbrood virus of the honey bee: An insect picorna-like virus," *J. Gen. Virol.*, vol. 80, no. 6, pp. 1541–1549, 1999, doi: 10.1099/0022-1317-80-6-1541.
- [61] L. Bailey, A. J. Gibbs, and R. D. Woods, "Sacbrood virus of the larval honey bee (*Apis mellifera* Linnaeus)," *Virology*, vol. 23, no. 3, pp. 425–429, 1964, doi: 10.1016/0042-6822(64)90266-1.
- [62] B. Arıcı, "Tokat ve Ordu İllerindeki Bal Arılarında (*Apis mellifera* L.) Deforme Kanat Virüsü (DWV) ve Tulumsuz Yavru Çürüklüğü Virüsü (SBV) Varlığının Araştırılması," vol. 2, no. 1, pp. 1–8, 2009.
- [63] J. R. de Miranda, G. Cordon, and G. Budge, "The Acute bee paralysis virus-Kashmir bee virus-Israeli acute paralysis virus complex," *J. Invertebr. Pathol.*, vol. 103, no. SUPPL. 1, pp. S30–S47, 2010, doi: 10.1016/j.jip.2009.06.014.
- [64] J. R. de Miranda *et al.*, "Complete nucleotide sequence of Kashmir bee virus and comparison with acute bee paralysis virus," *J. Gen. Virol.*, vol. 85, no. 8, pp. 2263–2270, 2004, doi: 10.1099/vir.0.79990-0.
- [65] M. F. Allen and B. V. Ball, "Characterisation and serological relationships of strains of Kashmir bee virus," *Ann. Appl. Biol.*, vol. 126, no. 3, pp. 471–484, 1995, doi: 10.1111/j.1744-7348.1995.tb05382.x.
- [66] G. E. Budge *et al.*, "Chronic bee paralysis as a serious emerging threat to honey bees," *Nat. Commun.*, vol. 11, no. 1, pp. 1–7, 2020, doi: 10.1038/s41467-020-15919-0.
- [67] A. Güller, M. Usta, G. Çakar, and Z. Kurt, "Molecular characterization of Deformed wing viruses identified in honeybee (*Apis mellifera* L.) colonies in Erzincan province of Turkey," *Eur. J. Sci. Technol.*, vol. 2021, no. 27, pp. 186–192, 2021, doi: 10.31590/ejosat.951040.
- [68] Ş. Kızıltepe, R. Akpınar, A. Akman, S. Kaya, and S. N. Çelik, "İğdır İlinde Görülen Arı Kolonisi Kayıplarında Viral ve Paraziter Etkenlerin Rolü," *İğdır Üniversitesi Fen Bilim. Enstitüsü Derg.*, vol. 13, no. 2, pp. 858–871, 2023, doi: 10.21597/jist.1184308.
- [69] Y. Yıldırım, "Bal Arılarının Viral Hastalıkları," *Cukurova Univ. Agric. Fac.*, vol. 1, no. 35, pp. 57–66, 2020, doi: 10.36846/cjafs.2020.18.
- [70] A. Balkaya, A. Güller, M. Usta, and Z. Kurt, "Prevalence of Deformed wing virus and Chronic bee paralysis virus in Honey Bee Colonies of Bingöl province, Turkey," *İğdır Üniversitesi Fen Bilim. Enstitüsü Derg.*, vol. 13, no. 1, pp. 44–53, 2023, doi: 10.21597/jist.1059418.
- [71] S. Gisder and E. Genersch, "Direct Evidence for Infection of Varroa destructor Mites with the Bee-Pathogenic Deformed Wing Virus Variant B, but Not Variant A, via Fluorescence In Situ Hybridization Analysis," *J. Virol.*, vol. 95, no. 5, 2021, doi: 10.1128/jvi.01786-20.
- [72] F. S. de Souza, M. H. Allsopp, and S. J. Martin, "Deformed wing virus prevalence and load in honeybees in South Africa," *Arch. Virol.*, vol. 166, no. 1, pp. 237–241, 2021, doi: 10.1007/s00705-020-04863-5.
- [73] J. L. Kevill, K. C. Stainton, D. C. Schroeder, and S. J. Martin, "Deformed wing virus variant shift from 2010 to 2016 in managed and feral UK honey bee colonies," *Arch. Virol.*, vol. 166, no. 10, pp. 2693–2702, 2021, doi: 10.1007/s00705-021-05162-3.
- [74] M. Gürçay and M. A. Kutlu, "Important Viruses of Honey Bees," *Int. J. Food, Agric. Anim. Sci.*, vol. 2, no. 2, pp. 29–41, 2022.
- [75] S. Bhatia, S. S. Baral, C. Vega Melendez, E. Amiri, and O. Rueppell, "Comparing survival of Israeli acute paralysis virus infection among stocks of U.S. honey bees," *Insects*, vol. 12, no. 1, pp. 1–13, 2021, doi: 10.3390/insects12010060.
- [76] L. Bailey, "The Epidemiology and Control of Nosema Disease of the Honey-Bee," *Ann. Appl. Biol.*, vol. 43, no. 3, pp. 379–389, 1955, doi: 10.1111/j.1744-7348.1955.tb02488.x.
- [77] Y. Chen *et al.*, "Asymmetrical coexistence of Nosema ceranae and Nosema apis in honey bees," *J. Invertebr. Pathol.*, vol. 101, no. 3, pp. 204–209, 2009, doi: 10.1016/j.jip.2009.05.012.
- [78] R. Galajda, A. Valenčáková, M. Sučík, and P. Kandrácová, "Nosema disease of European honey bees," *J. Fungi*, vol. 7, no. 9, 2021, doi: 10.3390/jof7090714.
- [79] D. L. Miller, E. A. Smith, and I. L. G. Newton, "A bacterial symbiont protects honey bees from fungal disease," *MBio*, vol. 12, no. 3, pp. 2–7, 2021, doi: 10.1128/mBio.00503-21.
- [80] I. Fries, "Nosema Apis- A Parasite in the Honey Bee Colony," *Bee World*, vol. 74, no. 1, pp. 5–19, 1993, doi: 10.1080/0005772X.1993.11099149.
- [81] H. S. Abdulhay and M. I. Yonius, "Effects of diseases and pests on honey bee (*Apis mellifera*) in different parts in Baghdad city, Iraq," *Plant Arch.*, vol. 20, pp. 220–223, 2020.
- [82] I. Fries *et al.*, "Standard methods for Nosema research," *J. Apic. Res.*, vol. 52, no. 1, pp. 1–28, 2013, doi: 10.3896/IBRA.1.52.1.14.
- [83] D. Y. Kim *et al.*, "The Ascosphaera Apis Infection (Chalkbrood Disease) Alters the Gut Bacteriome Composition of the Honeybee," *Pathogens*, vol. 12, no. 5, p. 734, May 2023, doi: 10.3390/pathogens12050734.
- [84] İ. Seven and P. Tatlı Seven, "Teknik Arıcılıkta Kritik Bakım ve Besleme Uygulamaları," *Fırat Üniversitesi Sağlık Bilim. Vet. Derg.*, vol. 32, no. 2, pp. 147–154, 2018.
- [85] A. Zerek, "Hatay İli Arıcılık İşletmelerinde Nosema ve Amöbe Enfeksiyonlarının Yaygınlığı," *Turkish J. Agricultural Nat. Sciences*, vol. 9, no. 4, pp. 976–981, 2022.
- [86] D. R. Kugonza, "Africa under attack: a continent-wide mapping of pathogens, parasites and predators afflicting the hived honey bee Apis

- mellifera L.(Hymenoptera: Apidae).” *African J. Rural Dev.*, vol. 5, no. 2, pp. 1–27, 2021.
- [87] M. Rossi, S. R. Ott, and J. E. Niven, “Malpighamoeba infection compromises fluid secretion and P-glycoprotein detoxification in Malpighian tubules,” *Sci. Rep.*, vol. 10, no. 1, pp. 1–12, 2020, doi: 10.1038/s41598-020-72598-z.
- [88] N. Şahinler and A. Gül, “Investigation of Bee Diseases in Beekeeping Enterprises in Hatay Province,” *Uludağ Bee J.*, vol. 1, no. 5, pp. 27–31, 2005.
- [89] H. Şimşek, “Elazığ Yöresi Bal Arılarında Bazı Parazit ve Mantar Hastalıklarının Araştırılması,” pp. 123–126, 2005.
- [90] A. E. Borum and M. Ülgen, “Güney Marmara Bölgesinde Bal Arılarının Chalkbrood (*Ascosphaera Apis*) Enfeksiyonunda Predispozisyon Faktörleri Predisposing Factors for Chalkbrood (*Ascosphaera apis*) Infection in Honey Bees in Northwest Turkey,” *Arı Bilimi / Bee Sci.*, vol. 10, no. May, pp. 56–69, 2010.
- [91] K. A. Aronstein and K. D. Murray, “Chalkbrood disease in honey bees,” *J. Invertebr. Pathol.*, vol. 103, no. SUPPL. 1, pp. S20–S29, 2010, doi: 10.1016/j.jip.2009.06.018.
- [92] U. Lermi, “Bartın Yöresi Bal Arısı (*Apis Mellifera L.*) Zararlıları ve Hastalıkları,” vol. 2, no. 5, pp. 1–179, 2010.
- [93] A. B. Jensen, K. Aronstein, J. M. Flores, S. Vojvodic, M. A. Palacio, and M. Spivak, “Standard methods for fungal brood disease research,” *J. Apic. Res.*, vol. 52, no. 1, 2013, doi: 10.3896/IBRA.1.52.1.13.
- [94] J. B. Benoit, J. A. Yoder, D. Sammataro, and L. W. Zettler, “Mycoflora and fungal vector capacity of the parasitic mite varroa destructor (mesostigmata: Varroidae) in honey bee (hymenoptera: Apidae) colonies,” *Int. J. Acarol.*, vol. 30, no. 2, pp. 103–106, 2004, doi: 10.1080/01647950408684376.
- [95] A. M. Shehabeldine, A. H. Hashem, and A. I. Hasaballah, “Antagonistic effect of gut microbiota of the Egyptian honeybees, *Apis mellifera L.* against the etiological agent of Stonebrood disease,” *Int. J. Trop. Insect Sci.*, vol. 42, no. 2, pp. 1357–1366, 2022, doi: 10.1007/s42690-021-00654-w.
- [96] K. Foley, G. Fazio, A. B. Jensen, and W. O. H. Hughes, “The distribution of *Aspergillus* spp. opportunistic parasites in hives and their pathogenicity to honey bees,” *Vet. Microbiol.*, vol. 169, no. 3–4, pp. 203–210, 2014, doi: 10.1016/j.vetmic.2013.11.029.
- [97] M. N. Shoreit and M. M. K. Bagy, “Mycoflora associated with stonebrood disease in honeybee colonies in Egypt,” *Microbiol. Res.*, vol. 150, no. 2, pp. 207–211, 1995, doi: 10.1016/S0944-5013(11)80058-3.
- [98] A. Alizadeh and M. S. Mossadegh, “Stonebrood and some other fungi associated with *Apis florea* in Iran,” *J. Apic. Res.*, vol. 33, no. 4, pp. 213–218, 1994, doi: 10.1080/00218839.1994.11100874.
- [99] S. Vojvodic, A. B. Jensen, R. R. James, J. J. Boomsma, and J. Eilenberg, “Temperature dependent virulence of obligate and facultative fungal pathogens of honeybee brood,” *Vet. Microbiol.*, vol. 149, no. 1–2, pp. 200–205, 2011, doi: 10.1016/j.vetmic.2010.10.001.
- [100] A. Noël, Y. Le Conte, and F. Mondet, “Varroa destructor: How does it harm *Apis mellifera* honey bees and what can be done about it?,” *Emerg. Top. Life Sci.*, vol. 4, no. 1, pp. 45–57, 2020, doi: 10.1042/ETLS20190125.
- [101] J. Dittes, H. Aupperle-Lellbach, M. O. Schäfer, C. K. W. Mülling, and I. U. Emmerich, “Veterinary Diagnostic Approach of Common Virus Diseases in Adult Honeybees,” *Vet. Sci.*, vol. 7, no. 4, p. 159, Oct. 2020, doi: 10.3390/vetsci7040159.
- [102] A. J. Mohammed, K. Abdul, and R. Fhad, “Evaluation Of Oils And Extracts Of Some Natural Materials In The Management Of Varroa Jacobsoni Oudemans Mites On *Apis Mellifera* Honey Bees In Basrah Province,” *Volatiles Essent. Oils*, vol. 9, no. 1, pp. 561–574, 2022.
- [103] L. Aydin, “*Aethina tumida* (Small Hive Beetle; SHB) and *Tropilaelaps* spp. Mite; an emerging threat to Turkey Honey Bees,” *Ankara Univ. Vet. Fak. Derg.*, vol. 69, no. 3, pp. 347–354, 2022, doi: 10.33988/auvfd.1019154.
- [104] A. O. Girişgin, L. Aydin, and Y. E. Yörük, “Hypothetical study of Small Hive Beetle *Aethina tumida* Infestation in Honeybees, Risk Commodities and Probabilities for Its Introduction in Türkiye,” *Kafkas Univ. Vet. Fak. Derg.*, vol. 28, no. 6, pp. 733–737, 2022, doi: 10.9775/kvfd.2022.28027.
- [105] A. Nanetti, J. D. Ellis, I. Cardaio, and G. Cilia, “Detection of lotmaria passim, crithidia mellificae and replicative forms of deformed wing virus and kashmir bee virus in the small hive beetle (*Aethina tumida*),” *Pathogens*, vol. 10, no. 3, 2021, doi: 10.3390/pathogens10030372.
- [106] Z. A. Jamal et al., “Future expansion of small hive beetles, *Aethina tumida*, towards North Africa and South Europe based on temperature factors using maximum entropy algorithm,” *J. King Saud Univ. - Sci.*, vol. 33, no. 1, p. 101242, 2021, doi: 10.1016/j.jksus.2020.101242.
- [107] P. Neumann, J. S. Pettis, and M. O. Schäfer, “Quo vadis *Aethina tumida*? Biology and control of small hive beetles,” *Apidologie*, vol. 47, no. 3, pp. 427–466, 2016, doi: 10.1007/s13592-016-0426-x.
- [108] H. Hernández Torres, A. Georgievich Kirejtshuk, C. Núñez Vázquez, and O. García Martínez, “On *Aethina tumida* Murray (Coleoptera: Nitidulidae: Nitidulinae) in hives of *Apis mellifera* Linnaeus (Hymenoptera: Apidae) in Campeche, México,” *J. Apic. Res.*, vol. 62, no. 2, pp. 326–329, 2023, doi: 10.1080/00218839.2021.1889223.
- [109] A. Karahan, M. A. Kutlu, E. Zengin, and İ. Karaca, “Awareness of Beekeepers About the Small Hive Beetle [*Aethina tumida* (Murray , 1867) (Coleoptera : Nitidulidae)], Which is A Threat to Beekeeping in Turkey,” *BinBee Arı ve Doğal Ürünler Derg.*, vol. 1, no. January, pp. 19–28, 2022.
- [110] L. Wu, L. Li, Y. Xu, Q. Li, F. Liu, and H. Zhao, “Identification and characterization of CYP307A1 as a molecular target for controlling the small hive beetle, *Aethina tumida*,” *Pest Manag. Sci.*, vol. 79, no. 1, pp. 37–44, 2023, doi: 10.1002/ps.7146.
- [111] G. F. de Landa et al., “Pathogens Detection in the Small Hive Beetle (*Aethina tumida* (Coleoptera: Nitidulidae)),” *Neotrop. Entomol.*, vol. 50, no. 2, pp. 312–316, 2021, doi: 10.1007/s13744-020-00812-8.
- [112] A. C. Croce and F. Scolari, “Autofluorescent Biomolecules in Diptera: From Structure to Metabolism and Behavior,” *Molecules*, vol. 27, no. 14, pp. 1–27, 2022, doi: 10.3390/molecules27144458.
- [113] M. M. Keshlaf, H. B. Mirwan, S. Ghana, S. Mubrok, and T. Shaibi, “Prevalence of Varroa mites (*Varroa destructor* Anderson & Trueman) and bee lice (*Bruala coeca* Nitzsch) in honey bee (*Apis mellifera L.*) colonies in Libya,” *Open Vet. J.*, vol. 13, no. 7, pp. 834–838, 2023, doi: 10.5455/OVJ.2023.v13.i7.4.
- [114] K. Gratzner, K. Wakjira, S. Fiedler, and R. Brodschneider, “Challenges and perspectives for beekeeping in Ethiopia,” *Agron. Sustain.*

- Dev., vol. 41, no. 4, pp. 2–10, 2021, doi: 10.1007/s13593-021-00702-2.
- [115] T. H. Büscher *et al.*, “The exceptional attachment ability of the ectoparasitic bee louse *Braula coeca* (Diptera, Braulidae) on the honeybee,” *Physiol. Entomol.*, vol. 47, no. 2, pp. 83–95, 2022, doi: 10.1111/phen.12378.
- [116] M. Z. Sharif, X. Jiang, and S. M. Puswal, “Pests, parasitoids, and predators: Can they degrade the sociality of a honeybee colony, and be assessed via acoustically monitored systems,” *J. Entomol. ...*, vol. 8, no. 3, pp. 1248–1260, 2020.
- [117] M. Pietropaoli *et al.*, “Molecular Detection of *Acarapis woodi* Using Hive Debris as Innovative and Non-Invasive Matrix,” *Appl. Sci.*, vol. 12, no. 6, pp. 10–14, 2022, doi: 10.3390/app12062837.
- [118] Y. Sakamoto, T. Maeda, M. Yoshiyama, F. Konno, and J. S. Pettis, “Differential autogrooming response to the tracheal mite *Acarapis woodi* by the honey bees *Apis cerana* and *Apis mellifera*,” *Insectes Soc.*, vol. 67, no. 1, pp. 95–102, 2020, doi: 10.1007/s00040-019-00732-w.
- [119] C. M. Peixoto, M. E. Correia-Oliveira, and C. A. L. De Carvalho, “Current Status of *Acarapis woodi* Mite Infestation in Africanized Honey Bee *Apis mellifera* in Brazil,” *Florida Entomol.*, vol. 102, no. 4, pp. 775–777, 2019, doi: 10.1653/024.102.0416.
- [120] V. V. Stolbova, “Current state of *Acarapis Hirst* mites (Acariformes, Tarsonemidae) distribution and honeybees infestation in Russia,” *Ukr. J. Ecol.*, vol. 11, no. 1, pp. 291–298, 2021, doi: 10.15421/2021.
- [121] S. Takashima, Y. Ohari, and T. Itagaki, “The prevalence and molecular characterization of *Acarapis woodi* and *Varroa destructor* mites in honeybees in the Tohoku region of Japan,” *Parasitol. Int.*, vol. 75, no. December 2019, p. 102052, 2020, doi: 10.1016/j.parint.2020.102052.
- [122] I. Serrano, C. Verdial, L. Tavares, and M. Oliveira, “The Virtuous *Galleria mellonella* Model for Scientific Experimentation,” *Antibiotics*, vol. 12, no. 3, pp. 1–25, 2023, doi: 10.3390/antibiotics12030505.
- [123] S. S. Saikia, B. K. Borah, G. Baruah, Rokozeno, and M. K. Deka, “Characterization of the gut microbes of greater wax moth (*Galleria mellonella* Linnaeus) shows presence of potential polymer degraders,” *Folia Microbiol. (Praha)*, vol. 67, no. 1, pp. 133–141, 2022, doi: 10.1007/s12223-021-00925-6.
- [124] E. Özgör, “The effects of nosema apis and nosema ceranae infection on survival and phenoloxidase gene expression in *Galleria mellonella* (Lepidoptera: Galleriidae) compared to *Apis mellifera*,” *Insects*, vol. 12, no. 10, 2021, doi: 10.3390/insects12100953.
- [125] E. M. Hosni, A. A. Al-Khalaf, M. G. Nasser, H. F. Abou-Shaara, and M. H. Radwan, “Modeling the Potential Global Distribution of Honeybee Pest, *Galleria mellonella* under Changing Climate,” *Insects*, vol. 13, no. 5, 2022, doi: 10.3390/insects13050484.
- [126] A. A. Egelie, A. N. Mortensen, L. Barber, J. Sullivan, and J. D. Ellis, “Lesser Wax Moth *Achroia grisella* Fabricius (Insecta: Lepidoptera: Pyralidae),” *Entomol. Nematol. Univ. Florida*, pp. 1–4, 2015.
- [127] Y. Liu, G. Li, and J. Long, “The complete mitochondrial genome of small wax moth, *Achroia grisella* (Pyralidae: Galleriinae),” *Mitochondrial DNA Part B Resour.*, vol. 7, no. 5, pp. 738–740, 2022, doi: 10.1080/23802359.2022.2068984.
- [128] L. Díaz-García, A. Reid, J. Jackson-Camargo, and J. Windmill, “Directional passive acoustic structures inspired by the ear of *Achroia grisella*,” *Proc. Meet. Acoust.*, vol. 50, no. 1, pp. 1–3, 2022, doi: 10.1121/2.0001715.
- [129] Chandresh B. Solanki, Birari Vaishali V., Manishkumar J. Joshi, and Prithiv Raj V., “Enemies of Honey bees and their Management,” *Agric. FOOD e-Newsletter*, vol. 2, no. 4, pp. 56–58, 2020.
- [130] P. Sabatina, G. Umapathy, and P. A. Saravanan, “Parasitisation Potential Of Egg And Larval Parasitoids Against Lesser Wax Moth *Achroia grisella* F. (Lepidoptera: Pyralidae) Under Stored Condition,” *Uludağ Arıcılık Derg.*, vol. 23, no. 2, pp. 215–223, 2023, doi: 10.31467/uluaricilik.1353063.
- [131] E. A. Wallin, B. Kalinová, J. Kindl, E. Hedenström, and I. Valterová, “Stereochemistry of two pheromonal components of the bumblebee wax moth, *Aphomia sociella*,” *Sci. Rep.*, vol. 10, no. 1, pp. 1–7, 2020, doi: 10.1038/s41598-020-59069-1.
- [132] H. K. Sharma *et al.*, “Seasonal incidence, epidemiology and establishment of different pests and disease in laboratory reared *Bombus haemorrhoidalis* Smith,” *Int. J. Trop. Insect Sci.*, vol. 41, no. 4, pp. 2555–2564, 2021, doi: 10.1007/s42690-021-00435-5.
- [133] S. E. Schweiger, N. Beyer, A. L. Hass, and C. Westphal, “Pollen and landscape diversity as well as wax moth depredation determine reproductive success of bumblebees in agricultural landscapes,” *Agric. Ecosyst. Environ.*, vol. 326, no. April 2021, p. 107788, 2022, doi: 10.1016/j.agee.2021.107788.
- [134] L. Aydın and S. Özgür, “Bal Arılarında Bulunan Az Önemli Zararlı Artropodlar (Eklem Bacaklılar),” *Uludağ Arıcılık Derg.*, vol. 12, no. May, pp. 40–54, 2012.
- [135] K. Arian, H. Burhan, E. Sahin, and F. Sen, “A sensitive, fast, selective, and reusable enzyme-free glucose sensor based on monodisperse AuNi alloy nanoparticles on activated carbon support,” *Chemosphere*, vol. 291, p. 132718, Mar. 2022, doi: 10.1016/j.chemosphere.2021.132718.
- [136] F. Karimi *et al.*, “Efficient green photocatalyst of silver-based palladium nanoparticles for methyle orange photodegradation, investigation of lipid peroxidation inhibition, antimicrobial, and antioxidant activity,” *Food Chem. Toxicol.*, vol. 169, p. 113406, Nov. 2022, doi: 10.1016/j.fct.2022.113406.
- [137] F. Göl, A. Aygün, A. Seyrankaya, T. Gür, C. Yenikaya, and F. Şen, “Green synthesis and characterization of *Camellia sinensis* mediated silver nanoparticles for antibacterial ceramic applications,” *Mater. Chem. Phys.*, vol. 250, p. 123037, Aug. 2020, doi: 10.1016/j.matchemphys.2020.123037.
- [138] R. Nagraik, A. Sharma, D. Kumar, S. Mukherjee, F. Sen, and A. P. Kumar, “Amalgamation of biosensors and nanotechnology in disease diagnosis: Mini-review,” *Sensors Int.*, vol. 2, p. 100089, 2021, doi: 10.1016/j.sintl.2021.100089.
- [139] Y. Wu *et al.*, “Hydrogen generation from methanolysis of sodium borohydride using waste coffee oil modified zinc oxide nanoparticles and their photocatalytic activities,” *Int. J. Hydrogen Energy*, vol. 48, no. 17, pp. 6613–6623, Feb. 2023, doi: 10.1016/j.ijhydene.2022.04.177.
- [140] K. Arian, H. Burhan, R. Bayat, and F. Sen, “Glucose nano biosensor with non-enzymatic excellent sensitivity prepared with nickel–

- cobalt nanocomposites on f-MWCNT,” *Chemosphere*, vol. 291, p. 132720, Mar. 2022, doi: 10.1016/j.chemosphere.2021.132720.
- [141] H. Karimi-Maleh, K. Cellat, K. Arıkan, A. Savk, F. Karimi, and F. Şen, “Palladium–Nickel nanoparticles decorated on Functionalized-MWCNT for high precision non-enzymatic glucose sensing,” *Mater. Chem. Phys.*, vol. 250, p. 123042, Aug. 2020, doi: 10.1016/j.matchemphys.2020.123042.
- [142] E. Demir, A. Savk, B. Sen, and F. Sen, “A novel monodisperse metal nanoparticles anchored graphene oxide as Counter Electrode for Dye-Sensitized Solar Cells,” *Nano-Structures & Nano-Objects*, vol. 12, pp. 41–45, Oct. 2017, doi: 10.1016/j.nanoso.2017.08.018.
- [143] A. Aygun, G. Sahin, R. N. E. Tiri, Y. Tekeli, and F. Sen, “Colorimetric sensor based on biogenic nanomaterials for high sensitive detection of hydrogen peroxide and multi-metals,” *Chemosphere*, vol. 339, p. 139702, Oct. 2023, doi: 10.1016/j.chemosphere.2023.139702.
- [144] B. Sen, S. Kuzu, E. Demir, T. Onal Okyay, and F. Sen, “Hydrogen liberation from the dehydrocoupling of dimethylamine–borane at room temperature by using novel and highly monodispersed RuPtNi nanocatalysts decorated with graphene oxide,” *Int. J. Hydrogen Energy*, vol. 42, no. 36, pp. 23299–23306, Sep. 2017, doi: 10.1016/j.ijhydene.2017.04.213.
- [145] “Front Matter,” in *Nanomaterials for Direct Alcohol Fuel Cells*, Elsevier, 2021, pp. i–ii. doi: 10.1016/B978-0-12-821713-9.09990-X.
- [146] H. Goksu, Y. Yıldız, B. Çelik, M. Yazici, B. Kilbas, and F. Sen, “Eco-friendly hydrogenation of aromatic aldehyde compounds by tandem hydrogenation of dimethylamine-borane in the presence of a reduced graphene oxide furnished platinum nanocatalyst,” *Catal. Sci. Technol.*, vol. 6, no. 7, pp. 2318–2324, 2016, doi: 10.1039/C5CY01462J.
- [147] N. Lolak, E. Kuyuldar, H. Burhan, H. Goksu, S. Akocak, and F. Sen, “Composites of Palladium–Nickel Alloy Nanoparticles and Graphene Oxide for the Knoevenagel Condensation of Aldehydes with Malononitrile,” *ACS Omega*, vol. 4, no. 4, pp. 6848–6853, Apr. 2019, doi: 10.1021/acsomega.9b00485.
- [148] F. Şen and G. Gökağaç, “Improving Catalytic Efficiency in the Methanol Oxidation Reaction by Inserting Ru in Face-Centered Cubic Pt Nanoparticles Prepared by a New Surfactant, tert -Octanethiol,” *Energy & Fuels*, vol. 22, no. 3, pp. 1858–1864, May 2008, doi: 10.1021/ef700575t.
- [149] Z. Ozturk, F. Sen, S. Sen, and G. Gokagac, “The preparation and characterization of nano-sized Pt–Pd/C catalysts and comparison of their superior catalytic activities for methanol and ethanol oxidation,” *J. Mater. Sci.*, vol. 47, no. 23, pp. 8134–8144, Dec. 2012, doi: 10.1007/s10853-012-6709-3.
- [150] F. Sen, A. A. Boghossian, S. Sen, Z. W. Ulissi, J. Zhang, and M. S. Strano, “Observation of Oscillatory Surface Reactions of Riboflavin, Trolox, and Singlet Oxygen Using Single Carbon Nanotube Fluorescence Spectroscopy,” *ACS Nano*, vol. 6, no. 12, pp. 10632–10645, Dec. 2012, doi: 10.1021/nn303716n.
- [151] E. Erken, Y. Yıldız, B. Kilbaş, and F. Şen, “Synthesis and Characterization of Nearly Monodisperse Pt Nanoparticles for C 1 to C 3 Alcohol Oxidation and Dehydrogenation of Dimethylamine-borane (DMAB),” *J. Nanosci. Nanotechnol.*, vol. 16, no. 6, pp. 5944–5950, Jun. 2016, doi: 10.1166/jnn.2016.11683.
- [152] B. Demirkan *et al.*, “Palladium supported on polypyrrole/reduced graphene oxide nanoparticles for simultaneous biosensing application of ascorbic acid, dopamine, and uric acid,” *Sci. Rep.*, vol. 10, no. 1, p. 2946, Feb. 2020, doi: 10.1038/s41598-020-59935-y.
- [153] B. Demirkan *et al.*, “Composites of Bimetallic Platinum-Cobalt Alloy Nanoparticles and Reduced Graphene Oxide for Electrochemical Determination of Ascorbic Acid, Dopamine, and Uric Acid,” *Sci. Rep.*, vol. 9, no. 1, p. 12258, Aug. 2019, doi: 10.1038/s41598-019-48802-0.
- [154] S. Ertan, F. Şen, S. Şen, and G. Gökağaç, “Platinum Nanocatalysts Prepared With Different Surfactants for C1–C3 Alcohol Oxidations and Their Surface Morphologies by AFM,” *J. Nanoparticle Res.*, vol. 14, no. 6, p. 922, Jun. 2012, doi: 10.1007/s11051-012-0922-5.
- [155] B. Şen, A. Aygün, A. Savk, S. Akocak, and F. Şen, “Corrigendum to ‘Bimetallic palladium–iridium alloy nanoparticles as highly efficient and stable catalyst for the hydrogen evolution reaction’ [Int J Hydrogen Energy 43 (2018) 20183–20191],” *Int. J. Hydrogen Energy*, vol. 46, no. 39, p. 20792, Jun. 2021, doi: 10.1016/j.ijhydene.2021.04.166.
- [156] B. Şen, A. Aygün, T. O. Okyay, A. Şavk, R. Kartop, and F. Şen, “Monodisperse Palladium Nanoparticles Assembled on Graphene Oxide With the High Catalytic Activity and Reusability in the Dehydrogenation of Dimethylamine Borane,” *Int. J. Hydrogen Energy*, vol. 43, no. 44, pp. 20176–20182, Nov. 2018, doi: 10.1016/j.ijhydene.2018.03.175.
- [157] F. Şen and G. Gökağaç, “Pt Nanoparticles Synthesized With New Surfactants: Improvement in C1–C3 Alcohol Oxidation Catalytic Activity,” *J. Appl. Electrochem.*, vol. 44, no. 1, pp. 199–207, Jan. 2014, doi: 10.1007/s10800-013-0631-5.
- [158] F. Şen, G. Gökağaç, and S. Şen, “High performance Pt nanoparticles prepared by new surfactants for C1 to C3 alcohol oxidation reactions,” *J. Nanoparticle Res.*, vol. 15, no. 10, p. 1979, Oct. 2013, doi: 10.1007/s11051-013-1979-5.
- [159] B. Sen, B. Demirkan, A. Şavk, S. Karahan Gülbay, and F. Sen, “Trimetallic PdRuNi Nanocomposites Decorated on Graphene Oxide: A Superior Catalyst for the Hydrogen Evolution Reaction,” *Int. J. Hydrogen Energy*, vol. 43, no. 38, pp. 17984–17992, Sep. 2018, doi: 10.1016/j.ijhydene.2018.07.122.
- [160] R. Ayrancı *et al.*, “Enhanced optical and electrical properties of PEDOT via nanostructured carbon materials: A comparative investigation,” *Nano-Structures & Nano-Objects*, vol. 11, pp. 13–19, Jul. 2017, doi: 10.1016/j.nanoso.2017.05.008.
- [161] M. B. Askari, P. Salarizadeh, A. Di Bartolomeo, and F. Şen, “Enhanced Electrochemical Performance of MnNi 2 O 4 /rGO Nanocomposite as Pseudocapacitor Electrode Material and Methanol Electro Oxidation Catalyst,” *Nanotechnology*, vol. 32, no. 32, p. 325707, Aug. 2021, doi: 10.1088/1361-6528/abfded.
- [162] R. Ayrancı, G. Başkaya, M. Güzel, S. Bozkurt, F. Şen, and M. Ak, “Carbon Based Nanomaterials for High Performance Optoelectrochemical Systems,” *ChemistrySelect*, vol. 2, no. 4, pp. 1548–1555, Feb. 2017, doi: 10.1002/slct.201601632.
- [163] S. Günbatar, A. Aygun, Y. Karataş, M. Gülcan, and F. Şen, “Carbon-Nanotube-Based Rhodium Nanoparticles as Highly-Active Catalyst for Hydrolytic Dehydrogenation of Dimethylamineborane at Room Temperature,” *J. Colloid Interface Sci.*, vol. 530, pp. 321–327, Nov. 2018, doi: 10.1016/j.jcis.2018.06.100.
- [164] P. Taslimi *et al.*, “Pyrazole[3,4-d]pyridazine derivatives: Molecular docking and explore of acetylcholinesterase and carbonic anhydrase

- enzymes inhibitors as anticholinergics potentials,” *Bioorg. Chem.*, vol. 92, p. 103213, Nov. 2019, doi: 10.1016/j.bioorg.2019.103213.
- [165] F. A. Unal, S. Ok, M. Unal, S. Topal, K. Cellat, and F. Şen, “Synthesis, Characterization, and Application of Transition Metals (Ni, Zr, and Fe) Doped TiO₂ Photoelectrodes for Dye-Sensitized Solar Cells,” *J. Mol. Liq.*, vol. 299, p. 112177, Feb. 2020, doi: 10.1016/j.molliq.2019.112177.
- [166] H. Göksu, Y. Yıldız, B. Çelik, M. Yazıcı, B. Kılbaş, and F. Şen, “Highly Efficient and Monodisperse Graphene Oxide Furnished Ru/Pd Nanoparticles for the Dehalogenation of Aryl Halides via Ammonia Borane,” *ChemistrySelect*, vol. 1, no. 5, pp. 953–958, Apr. 2016, doi: 10.1002/slct.201600207.
- [167] J. T. Abrahamson *et al.*, “Excess Thermopower and the Theory of Thermopower Waves,” *ACS Nano*, vol. 7, no. 8, pp. 6533–6544, Aug. 2013, doi: 10.1021/nn402411k.
- [168] B. Şahin *et al.*, “Cytotoxic effects of platinum nanoparticles obtained from pomegranate extract by the green synthesis method on the MCF-7 cell line,” *Colloids Surfaces B Biointerfaces*, vol. 163, pp. 119–124, Mar. 2018, doi: 10.1016/j.colsurfb.2017.12.042.
- [169] N. Korkmaz *et al.*, “Biogenic Silver Nanoparticles Synthesized Via Mimosa Elengi Fruit Extract, a Study on Antibiofilm, Antibacterial, and Anticancer Activities,” *J. Drug Deliv. Sci. Technol.*, vol. 59, p. 101864, Oct. 2020, doi: 10.1016/j.jddst.2020.101864.
- [170] F. Gulbagca, A. Aygün, M. Gülcan, S. Ozdemir, S. Gonca, and F. Şen, “Green Synthesis of Palladium Nanoparticles: Preparation, Characterization, and Investigation of Antioxidant, Antimicrobial, Anticancer, and DNA Cleavage Activities,” *Appl. Organomet. Chem.*, vol. 35, no. 8, Aug. 2021, doi: 10.1002/aoc.6272.
- [171] R. N. E. Tiri, F. Gulbagca, A. Aygun, A. Cherif, and F. Sen, “Biosynthesis of Ag–Pt bimetallic nanoparticles using propolis extract: Antibacterial effects and catalytic activity on NaBH₄ hydrolysis,” *Environ. Res.*, vol. 206, p. 112622, Apr. 2022, doi: 10.1016/J.ENVRES.2021.112622.
- [172] W. J. M. Steyn, “Potential applications of nanotechnology in pavement engineering,” *J. Transp. Eng.*, vol. 135, no. 10, pp. 764–772, 2009, doi: 10.1061/(ASCE)0733-947X(2009)135:10(764).
- [173] R. Hussain *et al.*, “Nano-managing silver and zinc as bio-conservational approach against pathogens of the honey bee,” *J. Biotechnol.*, vol. 365, pp. 1–10, Mar. 2023, doi: 10.1016/j.jbiotec.2023.01.009.
- [174] S. El-Sayied Ali *et al.*, “Exploring bee venom and silver nanoparticles for controlling foulbrood pathogen and enhancing lifespan of honeybees,” *Sci. Rep.*, vol. 14, no. 1, p. 19013, Aug. 2024, doi: 10.1038/s41598-024-67515-7.