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The Presence and Distribution of Nosemosis Disease in Turkey

Türkiye'de Nosemosis Hastalığı'nın Varlığı ve Dağıtımı

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Abstract

Nosemosis is one of the most important bee diseases causing economic losses in beekeeping, which is one of the significant reasons for Colony Collapse Disorder (CCD) in the world. *Nosemaapis* and *Nosemaceranae*, the microsporidian species, are the main causative agents of nosemosis in honey bees worldwide. This disease causes digestive system disorders, a decrease in the average life of bees and colony losses. In this review, the general characteristics of nosemosis disease, and information about the situation in Turkey are given.

Keywords: Honey bees, Microsporidia, Nosemosis, *Nosema apis*, *Nosema ceranae*, Turkey

Özet

Nosemosis hastalığı dunyada önemli ekonomik kayıplara neden olan Koloni Çökme Bozukluğu'nun en önemli nedenlerinden biri olarak kabul edilen arı hastalıklarından birisidir. *Nosemaapis* ve *Nosemaceranae*, (microsporidia türleri) nosemosis hastalığının iki etkenidir. Bu hastalık arıların sindirim sistemi bozukluklarına, ortalama ömrünün azalmasına ve koloni kayıplarına neden olur. Bu derleme makalesinde nosemosis hastalığının genel karakteristik özellikleri ve Türkiye'deki durumu hakkında detaylı bilgiler verilmektedir.

Anahtar kelimeler: Bal arısı, Microsporidia, Nosemosis, *Nosema apis*, *Nosema ceranae*, Türkiye

Abbreviations: CCD, Colony Collapse Disorder

1. INTRODUCTION

Turkey has great potential for beekeeping with some features such as climate and wealth of flora. The Beekeeping sector increases the importance of the economy due to these positive factors day by day. According to TUIK, 2017 dates, while Turkey placed in the second row after China with 5 million beehives, placed in the third row after China and Argentina with 82.003 tone honey production. Besides; while China placed in the first row with 46,4 kg, Turkey placed in the sixth row with 17,6 kg according to yield per colony (Tosun, 2012). The contradiction between hives number and honey production is due to bee diseases and pests prevalence which decreases the honey and larvae production, causing bee losses in winter and slow colony development in spring (Dagaroglu, 1999; Tosun &Yaman, 2016).

2. NOSEMOSIS

Microsporidia are obligate intracellular pathogens with a wide range of hosts that are nature infecting all animal phyla commonly insects and other invertebrates (Chen et al., 2009a; Higes, Martin& Meana, 2006). The Phylum Microsporidia has 200 genera and more than 1300 species (Becnel, Takvorian & Cali, 2014). Nosemosis is one of the most important diseases causing economic losses in beekeeping, which is one of the significant reasons for Colony Collapse Disorder (CCD) in the world (Cox-foxter et al., 2007; Paxton, 2010).

Nosema apis and *Nosema ceranae*, the microsporidian species, are the main causative agents of nosemosis in honey bees worldwide (Chen et al., 2009b; Higes et al., 2006; Paxton, 2010; Williams, Shafer, Rogers, Shutler & Stewart, 2008a). *N. apis* was the historic species infecting *Apis mellifera* (Hymonoptera: Apidae) honey bees. However, probably early in this century, *N. ceranae* became an invasive parasite of *A. mellifera*, transferring from Asian honey bees *Apis cerana* (Chen & Wang, 2007; Fries, Martín, Meana, García-Palencia & Higes, 2006; Higes et al., 2006; Huang, Jiang,). In addition, two species can co-infect honey bees which results in the more virulent infection on the host (Paxton, Klee, Korpella & Fries, 2007). These disease factors cause infection in adult bees' intestines, decrease bee life and decrease the honey production capacity of honey bees (Malone & Gatehouse, 1998).

Studies carried out are that *N. ceranae* causes a high rate of colony loss along with severe disease symptoms, unlike *N. apis* (Paxton, 2010). Martín- Hernández et al. (2009) showed that the honey bee individuals infected by *N. ceranae* are able to multiply and spread more rapidly than *N. apis* in suitable environmental conditions. In addition, it has been determined that *N. ceranae* causes nutritional stress in worker bees and causes more deaths (Mayack & Naug, 2009; Naug & Gibbs, 2009; OIE, 2008). Studies on the distribution and environmental resistance of *N. ceranae* show how different it is from *N. apis*. (Fries, 2010).

The characteristic stage of nosemosis infection is the spore stage. The spore contains taxonomically important structures such as polar filament, polaroplast, nuclei, and posterior vacuole. Huang et al. (2007) reported that polar filament forms 20 - 23 spirals in nosemosis spores and polar filament consists of 4 layers and other characteristic factors belong to a typical nosema. Chen et al. (2009a) reported that *N. cerenea* created 18-21 polar filament coils. In Huang (2012) study, the number of coils of the polar filament in *N. apis* spores is 27-30; Higes et al. (2006) and Fries, Feng, Silva, Slemenda &Pieniazek (1996) reported that *N. apis* spores formed 30 polar filament coils, *N. ceranae* spores formed 20-23 polar filament coils. Suwannapong, Maksong, Seanbualuang & Benbow (2010) reported exospore thickness on the sports wall as 25 - 50 nm. Chen & Huang (2010) say that the differences between *N. apis* and *N. ceranae* are

limited by their size and number of polar filament coils. Likewise, Huang (2012) and Brenna Traver, Matthew, Williams, Richard & Fell (2012) reported in their study that nosemosis disease factors were similar except for the number of polar filaments. The development stages of *N. apis* and *N. ceranae* pathogens in host tissues are the same (Fries, 2010; Higes, Garcia-Palencia, Martín-Hernández & Meana, 2007; Chen vd., 2009a). Unlike this, Huang (2012) reported in his study that there may be morphological differences between vegetative stages. Both disease factors are similar in terms of sports morphology, the most important difference in the sport's internal structure is the difference between the number of rings made by the polar filament and the size of the spore. These differences are based on the fact that *N. ceranaae* sports size and the number of polar filament coils are relatively smaller than *N. apis*, but these differences are not sufficient for the characterization of these two disease factors at the species level.

2.1. Symptoms and Tissue Pathogeny

Nosemosis disease has few external symptoms (Bailey, 1967; OIE, 2008; Whitaker, Szalanski & Kence, 2011). The only external symptom that is difficult to detect is behavioral changes. Campbell, Kessler, Mayack & Naug (2010) reported that infected young bees exhibit behaviors of mature bees. The external symptoms of *N. apis* and *N. ceranae* pathogens, which are the two factors of Nosemosis disease, are not very different from each other (Huang, 2012). As the symptoms of this disease, especially in the first months of spring, findings such as the presence of brown stools in front of the hives and honeycombs, the presence of diseased or dead adults at the entrance of the hive, separation of the wings, swelling of the abdomen, not flying and crawling on the ground are accepted (Bailey, 1967; Uygur & Giriskin, 2008; OIE, 2008). *N. ceranae* shows fewer symptoms than *N. apis* pathogen. Therefore, it is very difficult to detect. These external symptoms are evaluated as a preliminary finding and give clues about the presence of the disease.

Light microscopy studies are carried out by examining the fresh tissues dissected directly and comparing the morphological differences in the tissues where the infection is found. Intestinal lumen and epithelium, which is yellow and white and light brown in places in a healthy host, is mildly white or off-white with nosemosis disease, and it is more swollen than healthy intestine (Tosun, 2012). It is determined by the fact that spore structures, which are the characteristic life stage of the microspore pathogen in the tissues of the host, break the light in their way and have a wide oval structure with approximately the same shape and dimensions.

Spores belonging to nosemosis pathogen, morphologically thin oval - shaped small and spore ends are seen as sharp and symmetry. Tosun (2012) determined that *N. ceranae* was 4.9 x 2.83 µm in fresh samples and 4.41 x 2.47 µm in dyed samples. Huang et al. (2007) measured the length of *N. ceranae* spores as 4.5 x 2.4 µm. Chen

et al. (2009a) reported that *N. ceranae* spores are 3.9 - 5.3 µm length and 2.0 - 2.5 µm width. The World Animal Health Organization OIE (2008) reported that *N. apis* spores were 5 - 7 µm length and 3 - 4 µm width and declared with these measurements *N. apis* spores bigger than the *N. ceranae*. Although there are records in the literature that *N. ceranae* spores are smaller than *N. apis* spores as the spore morphology of these two disease factors, the differences that these two disease factors show morphologically are insufficient to distinguish between these two species (Chen & Huang, 2010; Higes et al., 2006, 2007; Fries, 2010).

It is known that nosemosis infection intensely infects the intestinal tissue of honey bees and vegetative stages of the microspore pathogen occur in the intestinal tissue (Fries, 2010; Higes et al., 2006). Chen et al. (2009a) reported that nosemosis infection intensely infects the intestine and body cavity. Martín-Hernańdez et al. (2009) reported that both disease factors did not cause infection in Malpighian tubes and muscle tissues. Besides this information, there are reports in the literature that nosemosis spores of honey bees are detected in salivary glands and secretion cells and Malpighian tubes, adipose tissue and muscle tissue by various methods (Chen et al., 2009a; Klee et al., 2007; Somerville & Hornitzky, 2007). *N. ceranae*spores spread faster in host tissues than *N. apis* spores (Paxton et al., 2007; Martín-Hernańdez et al., 2009). Huang (2012) reported that nosemosis disease factors were similar in terms of tissues infected in the host.

Light and electron microscopy studies are sufficient for the detection of *Nosema* microsporidium, which is the cause of nosemosis infection in honey bees, at the level of genus (Chen et al., 2009a; Fries, 2010; Higes et al., 2007; Huang et al., 2007). This is the most important reason why it is thought that the only cause of nosemosis disease in honey bees in Europe and Asia for many years is *N. apis*. Studies in recent years have been carried out with molecular techniques and the presence of a second disease factor has been determined. It was revealed that *N. apis* records, which were previously defined by light and electron microscopy with the developed molecular techniques, were *N. ceranae*. Molecular characterization is required to determine which nosemosis disease is caused by these two factors in honey bees (Bourgeois, Rinderer, Beaman & Danka, 2010; Higes et al., 2006, 2007; Huang et al., 2007; Klee et al., 2007; OIE, 2008). Almost all of the studies on nosemosis infections detected in Turkey is the light microscopeespecially until 2010.

2.2. Transmission

It is known that the stools in front of the hive caused by the infected bees and the death of infected bees near the hive play an important role in the spread of nosemosis. In many studies, it has been reported that healthy worker bees make direct contact with *Nosema* spores while working to clean the feces in the flying board in front of

the hive. Also, there are many reports that *Nosema* spores are transported to other individuals in the hive after the contact of pollen bees with feces in front of the hive and infected bees (Brenna et al., 2012; Chen & Huang, 2010; Fries, 2010). Fries (1993) reported that feeding and defecation played an effective role in the spread of infection by *N. apis*, and again, Fries (2010) reported that the factor in the spread of *N. ceranae* in hives is unknown. Fenoy, Rueda, Higes, Martín-Hernandez & del Aguila (2009) reported that the honey wax melt in beekeeping and reused in the new season retain the infectivity of the pathogen spores and infect clean hives in the new honey season. With the precautions to be taken, the ways of infection can be cut and the speed of infection can be controlled.

2.3. Presence in the Honey Bee Colony

The number of detailed studies that determine which individuals in the colony occurred in infection studies conducted in Turkey is quite limited. Only Tosun (2012) determined that while worker bees were infected with *N. ceranae* infection, it was not found in male and queen bees. Chen et al. (2009b) and Somerville & Hornitzky (2007) said that nosemosis infection can cause infection in male bees as well as worker and queen bees, but the presence of infection in the colony individuals has not been reported in either study. Besides, Webster, Pomper, Hunt, Thacker & Jones (2004) detected the infection only in worker and queen bees, which are female individuals. Czekońska (2000) detected nosemosis infection only in female individuals. In the experimental study he conducted in the same study, he proved that the infection was transmitted from queen bees to worker bees. Webster, Thacker, Pomper, Lowe & Hunt (2008) in their study, nosemosis spores do not have vertical transmission like other microsporidia; reported no nosemosis in eggs, larvae, and pupae developing in infected queens. Martín-Hernańdez et al. (2009) reported that *N. ceranae* infection is more deadly than *N. apis* infection in worker bees. Malone, Gatehouse & Tregidga (2001) investigated the presence of *Nosema* infection in terms of the number of spores in beehives and bees in charge of collecting pollen and stated that nosemosis infection is different. Also, Brenna et al. (2012) stated that it did not show a significant difference.

2.4. Management

If nosemosis infection is not controlled, it may cause colonies to collapse, especially if the queen bee gets infected (Higes et al., 2008; Martín-Hernańdez, Meana, Prieto, Salvador, Garrido-Bailón & Higes, 2007). Today, the fight against this disease is mostly done in the form of chemical control. Fumagilin-B® (Medivet Pharmaceuticals Ltd.) is used extensively in the fight against nosemosis infection (Williams, Sampson, Shutler & Rogers, 2008b; Bourgeois et al., 2010; Fries, 2010). In addition, physical combat techniques, which are not preferred by beekeepers due to the difficulty of implementation and the need for intense labor, have the potential to be used in combating this disease. For example, the treatment of hive

materials with a temperature of 24 hours at 49 ºC ensures that *N. apis* infection is eliminated (Malone et al., 2001). Nosemosis disease can be easily detected by careful monitoring of symptoms. The most important of these symptoms is the presence of feces in front of the hive. Beekeepers can control the presence of the disease by taking the necessary precautions when they detect these external symptoms. Especially the humidity increases the amount of infection. Controlling the moisture in the hives by the beekeepers will affect the existence of the disease and the disease can be taken under control.

Besides the chemicals, which are widely used to control the infection, it is effective in reducing the presence of nosemosis infection in the measures taken by the beekeepers with their own experience. Among these, the methods used to decrease the moisture content in the hive come first. In addition, it prevents the spread of a possible nosemosis infection in the collection of the dead in front of the hive and cleaning the feces in front of the hive. The presence of *Nosema* spores can be reduced with the method of sterilization for flame cleaning in the hive for spring cleaning in the hive or during the storage process.

2.5. Nosemosis in Turkey

In 1986, the first identification of *N. apis* infection was done in laboratory of "Turkiye Kalkınma Vakfı Arı Hastalıkları" (Tutkun &Inci 1992). In 1988, atotal of 15600 worker bees from 312 apiaries were inspected on light microscopy and the averageinfection rate was reported as 26.4% by Kutlu& Kaftanoglu (1990). In this study, reported that *N. apis* was found in Mugla (31.3%), Adana (29.8%), Dalaman (29.6%), Aydın (28,.6%), Datca (25.7%), Milas (25.0%), Fethiye (23.8%), Koycegiz (23.3%) and Marmaris (20.5%) respectively.

Between 1988 and 1989, Basar (1990) investigated *N. apis* infections of honey bees in Trakya region, Mugla and Istanbul provinces. A total of 9590 worker bees from 126 hives were examined on light microscopy by Basar (1990). The intensity of *Nosema* spores per bee was between 0.5 million and 16 million and the maximum level of infection was reached at spring and winter in the same study. Additionally, the highestintensity ofinfectionwas reported in Trakya region. In another study, Keskin, Basar & Saracbası (1996) examined 7820 honey beein the sameyear (1988-1989) and in the same localities (Trakya regions, Mugla and Istanbul provinces) with Basar (1990). Additionally, Keskin et al. (1996) reported that the highest density of *Nosema* infection was observed from April to November.

In 1999, Ozbilgin, Alatas, Balkan, Ozturk & Karaca (1999) reported that*Nosema*infection ratewas 2% for the Aegean Region of Turkey.

In 2001, the nosemosis infection research reported by Ozkırım &Keskin (2001) in Anzer locality has been regarded as one of the most important studies for Turkey. Because the "Anzer honey" which is produced in Anzer locality of Rize province is the most famous and expensive

in Turkey. In that study, Ozkırım & Keskin (2001) reported that *N. apis* infection was observed on light microscopy in Anzer, but they did not report infection rate in their study. In another study, Aydın, Gulegen &Cetinbas (2001a, 2001b) found that the prevalence of *N. apis* spores was 26.4% in Bursa province, and 26.25% in the South Marmara Region of Turkey. Additionally, Cengiz & Genc (2001) reported that nosemosis infection rate was 4.48% in Erzurum according to a survey conducted. In another study conductedin the same year, the prevalence of nosemosis infection was reported as 4% in the center of Elazıg, 4% in Baskil and 10% Sivrice localities in a study conducted in Elazig province by Simsek, Dilgin & Gultekin (2001). Kutlu & Gazioglu (2008) reported that a total of 47 of 122 hives were infected with nosemosis which infection rates varied from 52.9% to 25% and the average contamination rate 38.5% in Bingol provinces.

In 2002, nosemosis infection rate was reported for the Black Sea Region of Turkey in beekeeping apiaries was 30.95% (Yasar, Guler, Yesiltas, Bulut & Gokce, 2002). The presence of nosemosis infection was determined by Aydın, Cakmak, Gulegen & Korkut (2003) with a survey conducted with 50 beekeepers in the Bursa and Yalova provinces of South Marmara Region in March 2002.

In 2003, Cakmak, Aydın, Seven & Korkut (2003a) and Cakmak, Aydın & Gulegen (2003b) reported nosemosis infection rate as 24% in 217 hives in the South Marmara Region. In another study, Kutlu & Ekmen (2003) inspected 1220 worker bees from 122 hives in Bingol provinces and reported that nosemosis infection rate was between 25% and 52.9% (average 38.5%) in 2003.

In 2004, Topcu & Aslan (2004) observed *N. apis* infection in 54 of 343 (15.74%) honey bee hives which were examined in terms of nosemosis in the Kars province. In the same study, nosemosis infection rates were reported as 28.0% in Kagızman, 20.69% in Selim, 18.56% in Kars Center, 18.33% in Susuz, 15.79% in Digor, 13.04% in Arpacay, and 6.82% in Akyaka localities, and also no infection was recorded in Sarıkamıs locality by Topcu and Aslan (2004). Additionally, *N. apis*infection was found at the highest levelinMay-June in Kars (Topcu & Aslan 2004).

Furthermore, from the year 2002 to 2004,the percentage of Nosema infection wasreported as8.77% inElazıg province by Simsek (2005).

In 2005, Aydin, Cakmak, Gulegen &Wells (2005) reported that Nosema infection rate was identified on light microscopy as 60% of the apiaries sampled from seven regions in Turkey. Marmara and Black Sea Regions have higher infection rates than other regions in Turkey. There was no infection in the Southeast Anatolia Region. Additionally, the temperature was a significant factor in the presence of nosemosis disease. And also rainfall and humidity factors are more effective than temperature factors on nosemosis infection (Aydin et al., 2005). In

another study, the presence of nosemosis without specifying the species name was reported as an average of 6.5% in Edirne, Tekirdag, Kırklareli, Istanbul and Canakkale provinces in Trakya and Marmara Regions by Sıralı & Dogaroglu (2005). Soysal & Gurcan (2005) reported that 9% of beekeepers had apiaries infected with Nosema disease in their questionnaire study in Tekirdag in 2005.

In 2007, nosemosis infection rates varied from 25% to 54.16% (average rate of 42.45%) in 68 of 147 apiaries that reported by Kutlu & Gazioglu (2008). Besides reported that nosemosis illness showed an increase of 10.25% in 2007 compared to 2001.

Between the years 2003 and 2007, Giray, Kence, Oskay, Doke & Kence (2010) reported that Nosema infection *(N. apis* or *N. ceranae* is not specified) was accounted for 9% of colony losses among all causes in Turkey especially from 2006 to 2007.

In 2009, the queen honey beesinfected with*Nosema*sp. was reported for the first time by Muz & Muz (2009) in Hatay. Yalcınkaya, Keskin & Ozkırım(2009) investigate 3880 adult honeybee from Adana province and 3520 adult honeybee from Hatay province, and published nosemosis (without the name ofthe species) infection as 12.97% in 2009. Gul & Kutlu (2009) investigated the presence of Nosema disease in six localities in Bingöl province and reported *Nosema* infection rate as 8.41% in 2009.

Between the years 2007 and 2009, the first study aboutthe moleculardiagnosis of nosemosis was reported by Muz, Girisgin, Muz & Aydın (2010). In that study, Muz et al. (2010) reported that Hatay province had 89% *N. cerana* and 11% *N. apis* infections, in addition to the Marmara region were found to be 84% *N. cerana* and 16% *N. apis* infections.

From the year 2010, many scientists have begun to use molecular techniques to determine the factor (*N. apis* or *N. ceranae*) that causes nosemosis disease in Turkey. As mentionedabove, the first*N. ceranae*infection in honey bees in Turkey was detected from thespecimenscollected from the Marmara region between the years2007 and 2009by Muzet al.(2010). Utuk, Piskin & Kurt (2010) reported the presence of *N.ceranae*infection in Giresun and Sivas provinces in 2010. In that study, the infection rate was not reported butthe existence of *N. ceranae* was mentioned (Utuk et al. 2010). In the same year, Whitakeret al.(2011)reported the distribution of*N. ceranae* fromTurkey for the first time. The percentage ofnosemosis diseasewasdetermined as8.3% in Turkey by Whitakeret al.(2011) in 2010. In the same study, Whitakeret al.(2011)determined that the percentage ofinfection caused by*N. apis* was 4.7% in 4 of 20 provinces (Sivas, Izmir, Bitlis and Gaziantep), whilethe percentage ofinfection caused by*N. ceranae* was3.5% in 3 of 20 provinces (Artvin Hatay and Mugla) in Turkey in 2010. Any nosemosis infection was not observed in Gokceada locality in Canakkale province, Kırklareli, Bursa, Sakarya, Duzce, Giresun,

Ankara, Gaziantep, Adıyaman, Diyarbakır Batman, Sırnak and Erzincan provinces in 2010.

From 2006 to 2011, Utuk, Piskin, Girisgin, Selcuk & Aydın (2016) reported that *N. apis* infection as 6.25% in Cankırı province and 93.75% in Ankara, Bursa, Erzurum, Kayseri, Mugla, and Zonguldak provinces for Turkey.

From 2009 to 2011, *N. ceranae* was determined as the only factor of nosemosis in the Eastern Black Sea Region of Turkey with molecular techniques by Tosun (2012). A total of 5330 dead worker bees, 559 dead male bees and 4 dead queen bees collected from 20 different localities in Artvin, Rize, Trabzon, Giresun, Ordu, Gumushane and Bayburt provinces were examined for nosemosis and only worker bees were observed to be infected (Tosun, 2012). *N. ceranae* infection rates were reported as 4.72%, 15.28% and 21.23% in 2009, 2010 and 2011, respectively, the total infection was 20.59% and highest infection rates were observed in June and July (Tosun, 2012). Also, Tosun & Yaman (2016) reported that *N. ceranae* infectionwas affected bychangingtemperature and humidityfactors around the hives. Additionally, the humidity was more effective than the temperature factor on *N. ceranae* infection.

Between 2010 and 2011, Muz, Solmaz, Yaman & Karakavuk (2012) determined 10% of Nosema disease ofhives in winteringseason in Hatay province.

Between 2011 and 2012 Ivgın Tunca, Oskay, Gosterit & Tekin (2016) reported that *N. ceranae* infection observed in Izmir, Aydın, Mugla, Tekirdag, Kirklareli, Zonguldak, Artvin Isparta, Adana and Kırsehir range of 8.8-100% rates. The main point is that the article all samples were negative for *N. apis*.

In 2015 *N. ceranae* infection was reported with 3.28% in Ordu province by Guner, Erturk & Yaman (2019). Additionally, Oguz Karapınar, Dincer & Deger (2017) reported Nosema spp. Infection rate as 32.5% in Van Province.

From 2009 to 2016 Ozkırım, Shiesser & Keskin (2019) made research on the presence of nosemosis infection in 72 provinces of Turkey. They found three types of infection such as single *N. apis* infection, single *N. ceranae* infection and mixed infection with both species. *N. apis* infection rates reported as 16.3% in 2009, 8.8% in 2010, 21.7% in 2011, 29.2% in 2012, 20.5% in 2013 19.7% in 2014, 22.5% in 2015 and 22.3% in 2016. For *N. ceranae* 63.4% in 2009, 72.6% in 2010, 32.3% in 2011, 26.8% in 2012, 33.1% in 2013 34.2% in 2014, 28.5% in 2015 and 31.9% in 2016 rates were reported. Additionally, co-infection with both species 20.3% in 2009, 18.6% in 2010, 46% in 2011, 44% in 2012, 46.4% in 2013 46.1% in 2014, 49% in 2015 and 45.8% in 2016 reported in that study. According to the data in that study, especially winter conditions changed the rates of nosema infection levels in colonies.

3. RESULTS AND DISCUSSION

Turkey has a geographical location that connects Asia to Europe. Trade and globalization play an

important role in the rapid spread of nosemosis infection all over the world. In bees with nosemosis infection, the appearance is quite similar to the two disease factors in external symptoms such as intestinal and abdominal changes. The development stages of *N. apis* and *N. ceranae* pathogens in host tissues are the same. The differences between *N. apis* and *N.ceranae* are limited by the size of spores and the number of polar filament rings. Although there are records in the literature that *N. ceranae* spores are smaller than *N. apis* spores as the spore morphology of these two disease factors, the differences that these two disease factors show morphologically are not sufficient to characterize at the species level.

Light and electron microscopy studies are sufficient for the detection of *Nosema* microsporidium in the genus level, which is the cause of nosemosis infection in honey bees. For nosemosis, molecular characterization is required to determine to differentiate the disease factor in species level. In Turkey, this disease was mostly determined by looking at the spore morphology by light microscopy. In most of these studies, while there was no emphasis on the disease factor, only a few studies were accepted as *N. apis*. There are very few studies on whether the cause of nosemosis disease occurring in bee colonies in different regions of our country is *N. apis* or *N. ceranae.*

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