

## The relationship between the infection levels of *Nosema apis* parasites and epithelial cells of mid-gut of honeybee in Qena Government

Mohammed Z. Y. Aly<sup>1</sup>; Karem M. Mohanny<sup>2</sup>; Hoda S. Mohamedain<sup>1</sup>;  
Rasha A. S. Elnasr<sup>1</sup>

1-Zoology Department, Faculty of Science (Qena), South Vally University

2-Plant Protection Department, Faculty of Agriculture (Qena), South Vally University

### ABSTRACT

This study was conducted at Faculty of Agriculture, South Valley University, Qena Government, Egypt during the seasons 2011, between two hybrid of the Carniolian bee race and second hybrid of the Italian bee race to study the relationship between the infection levels of *Nosema apis* spores and epithelial cells of midgut of honeybee.

The results showed that there were high significant differences between the average numbers of spores; where the numbers of spores in Carniolian bee race higher than the Italian bee race (20467.26-11402.03), respectively during the study period.

On the other hand histological section showed that heavy infection lead to the disintegration of epithelial cell walls, striated border, and fragmentation of peritrophic membranes. Also, destruction of the muscle layer and basement membrane, while in the case of moderate cells infection appeared semi-decomposed compared to healthy cells.

The research recommends to intensify the care and treatment programs during months of heavy infection (January, February, March), in the southern states.

**Keywords:** Honeybee, Worker, Mid-gut, *Nosema apis*, spores, Histopathology

### INTRODUCTION

Nosemosis is the most widespread of adult bee diseases and causes significant economic losses to beekeepers worldwide. This disease was originally thought to be caused by a single *Nosema* sp., *Nosema apis* Zander, a microsporidian which has a range of effects on honey bee colonies and adult bee (Thomas *et al.* 2009). Transmission of *Nosema* in honey bee colonies is mainly via the fecal-oral route in which pathogens are spread by transferring feces of disease hosts to uninfected hosts via ingestion (Yanping *et al.* 2008). The spores germinate within the midgut and release polar tubes that transfer their sporoplasm into midgut epithelial cells where they generate more spores. Millions of new spores can be found inside a midgut bees, a few weeks after initial infection (Bailey and Ball, 1991) and the spores excreted with feces become new sources of infection in the colonies. Newly ingested spores were found closer to the apex of the epithelium as they moved toward the distal end.

The initial infection of the epithelial cells was restricted mainly to a very narrow area, at the posterior end of the midgut. Neither released sporoplasms in the midgut lumen, nor were polar tubes penetrating the host cell plasmalemma detected. The differences in the fine structure between the first and second meronts were minor (Graaf *et al.* 1994). The parasites grow and multiply producing wide varieties of histopathological modification and damage of infection parts. Tissue damage of bees

may produce bees dysentery, weakness and partial paralysis (Dyess and Wilson, 1978). Also, death as a result of starvation may occur (Muresan *et al.*, 1975).

The objective of the present work is to investigate the seasonal fluctuations in infection levels and histological studies on infected mid-gut.

## MATERIAL AND METHODS

The present study was carried out at Faculty of Agriculture, South Valley University, Qena Government, Egypt during the seasons 2011.

The hybrid of the carniolan bee race and second hybrid of the Italian bee race were chosen to start the planned experiment.

### 1-Prepared the bee colonies:

1-1 Twelve colonies hybrid of Carniolan bee race and Twelve colonies 2<sup>nd</sup> hybrid of Italian bee race (two group) were chosen for the work.

1-2 The colonies were divided into 2 groups of 12 colonies each. (Carniolan and Italian).

1-3 The 1<sup>st</sup> group was Carniolan (6 colony infection bees and 6 colony healthy).

1-4 The 2<sup>nd</sup> group was Italian (6 colony infection while 6 colony healthy).

1-5 Random samples of worker bees were collected from the central part of brood comb (nurse bees) of each colony (5 individuals) and that number was considered to represent the colony.

### 2-The process of preparing and the plan of the work:

#### 2.1. Counting of *Nosema* spores:

The alimentary canal of each 5-individual bee were taken out of its body by pulling the tip of the abdomen with a pair of forceps (Hassanein, 1952a). The ventriculus and the small intestine of each bee were macerated in 5 ml distilled water using a clean glass rod and mortar and pestle. A droplet of this suspension was placed onto a glass slide, and examined at X400 magnification under the light microscope for the presence of *Nosema apis* spores. A droplet of the same suspension was placed on a hemacytometer to determine the mean number of spores per 5-individual bee (in millions) from infected samples (Cantwell, 1970). Since this sample was known to be adequate for most purposes. Both percentages of infected bees and the mean number of spores/bee in each sample were estimated. A quantitative measure of levels of *Nosema* can be obtained using a hemacytometer, (an instrument used to count human blood cells).

To count, find the ruled area and focus the microscope on the spores so they are sharply defined.

To obtain a good average, count five blocks of 16 small squares. For more detail see Cantwell (1970).

#### 2.2. Technique for Histological Section:

The histological investigation was carried out on the alimentary system of healthy and *Nosema* infected worker honey bees (Elshemy, 1986). The alimentary canals of healthy and infection workers were fixed for 2 h in Alcoholic Bouin's solution which was prepared. The specimens were then dehydrated in an ascending alcohol series, cleared in Xylene and embedded in paraffin wax. Sections (5  $\mu$  thick) were cut and stained with Haematoxyline and Eosin. These sections were examined and photographed by using the microscope.

#### Statistical analysis

Statistical analysis of bioassay results for L.S.D. and F.0.05.

## RESULTS AND DISCUSSION

### Counting of *Nosema* spores

As shown in Table (1) and presented graphically in Fig (1) the average number of *Nosema* infection levels (mean no. of spores/bee) in nurse bees for two honey bee hybrids during the months were as follows:

The highest number of *Nosema* spores/bee was Carniolian bee race followed by Italian bee race.

Table 1: Average number of *Nosema* infection levels (mean no. of spores/bee) in nurse bees for two honey bee hybrids during the months of year 2011.

Sampling date		Carniolian	Italian	F 0.05	LSD
January	11	1.11x10 <sup>6</sup>	1.07 x10 <sup>6</sup>	-	-
	18	1.20 x10 <sup>6</sup>	1.23 x10 <sup>6</sup>	-	-
	25	1.28 x10 <sup>6</sup>	1.09 x10 <sup>6</sup>	-	-
	Mean	1.19 x10 <sup>6</sup>	1.13 x10 <sup>6</sup>	2.17ns	62769.26
February	1	1.27 x10 <sup>6</sup>	1.48 x10 <sup>6</sup>	-	-
	8	0.67 x10 <sup>6</sup>	0.64 x10 <sup>6</sup>	-	-
	15	1.21 x10 <sup>6</sup>	0.54 x10 <sup>6</sup>	-	-
	22	1.10 x10 <sup>6</sup>	0.83 x10 <sup>6</sup>	-	-
March	Mean	1.06 x10 <sup>6</sup>	0.87 x10 <sup>6</sup>	541.5***	22669.57
	1	1.11 x10 <sup>6</sup>	1.08 x10 <sup>6</sup>	-	-
	8	0.64 x10 <sup>6</sup>	0.52 x10 <sup>6</sup>	-	-
	14	0.73 x10 <sup>6</sup>	0.30 x10 <sup>6</sup>	-	-
April	22	0.20 x10 <sup>6</sup>	0.34 x10 <sup>6</sup>	-	-
	29	0.20 x10 <sup>6</sup>	0.15 x10 <sup>6</sup>	-	-
	Mean	0.58 x10 <sup>6</sup>	0.48 x10 <sup>6</sup>	13.5*	45339.15
May	5	308.33	46.67	-	-
	12	208.33	53.33	-	-
	19	353.33	170.00	-	-
	26	283.33	226.67	-	-
June	Mean	288.33	124.17	314.59***	1.68
	3	205.00	140.00	-	-
	10	171.67	280.00	-	-
	24	193.33	203.33	-	-
July	31	204.17	190.42	-	-
	Mean	193.54	203.44	540.85***	15.12
	7	111.67	135.00	-	-
	14	51.67	180.00	-	-
August	21	105.00	123.33	-	-
	29	128.33	139.17	-	-
	Mean	99.17	144.37	438.66***	1.68
Sampling date		Carniolian	Italian	F 0.05	LSD
September	5	105.00	41.67	-	-
	12	83.33	5.00	-	-
	19	28.33	3.33	-	-
	26	25.00	3.33	-	-
October	Mean	60.42	13.33	272.88***	2.51
	2	40.00	5.00	-	-
	9	58.33	26.67	-	-
	23	31.67	70.00	-	-
November	Mean	43.33	33.89	307.58***	4.83
	6	43.33	11.67	-	-
	13	15.00	12.62	-	-
	20	28.33	8.00	-	-
December	Mean	28.89	10.76	719.93***	1.87
	4	71.67	0.00	-	-
	18	56.67	0.00	-	-
	25	41.67	6.67	-	-
January	Mean	56.67	2.22	175.39***	1.13
	1	111.67	8.33	-	-
	8	158.33	13.33	-	-
	22	170.00	28.33	-	-
February	Mean	233.33	56.67	108.76	1.19
	Mean	168.33	26.67	-	-
	6	135.00	134.44	-	-
	13	251.67	133.89	-	-
March	20	250.00	140.00	-	-
	27	535.00	198.33	-	-
	Mean	292.92	151.67	309.94***	2.23
F 0.05		311.93***	933.47***	-	-
LSD		20467.26	11402.03	-	-

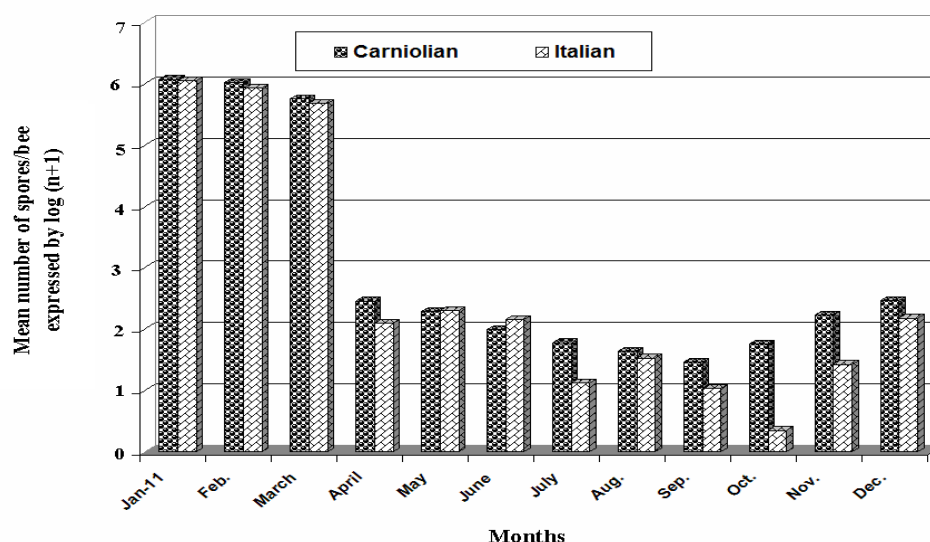


Fig. 1: Seasonal fluctuation of *Nosema apis* infection levels (mean no. of spores/bee) in nurse bees for two honey bee hybrids during the months of year 2011.

**Carniolian bee race:** The mean numbers of spores/bee were highest in the January, February, March and December ( $1.19 \times 10^6$ ,  $1.06 \times 10^6$ ,  $0.58 \times 10^6$  and 292.92 spores/infected bee), respectively followed by April, May, and November and June (288.33, 193.54, 168.33 and 99.17 spores/infected bee), respectively. The least number of spores/bee was in July, October, August and September (60.42, 56.67, 43.33 and 28.89 spores/infected bee), respectively.

**Italian bee race:** The mean numbers of spores/bee were highest in the January, February, and March ( $1.13 \times 10^6$ ,  $0.87 \times 10^6$  and  $0.48 \times 10^6$  spores/infected bee), respectively. Followed by May, December, June and April (203.44, 151.67, 144.37, and 124.17 spores/infected bee), respectively. The least number of spores/bee was in August, November, July, September and October (33.89, 26.67, 13.33, 10.76 and 2.22 spores/infected bee), respectively.

The statistical analysis showed that the differences in the mean number of spores/bee per colony for two honey bee hybrids (Carniolian and Italian) were high significant respectively during months.

As shown in Table (2) and presented graphically in Fig (2) the average number of *Nosema* spores/bee in nurse bees for two honey bee hybrids during seasons of year 2011 were as follows:

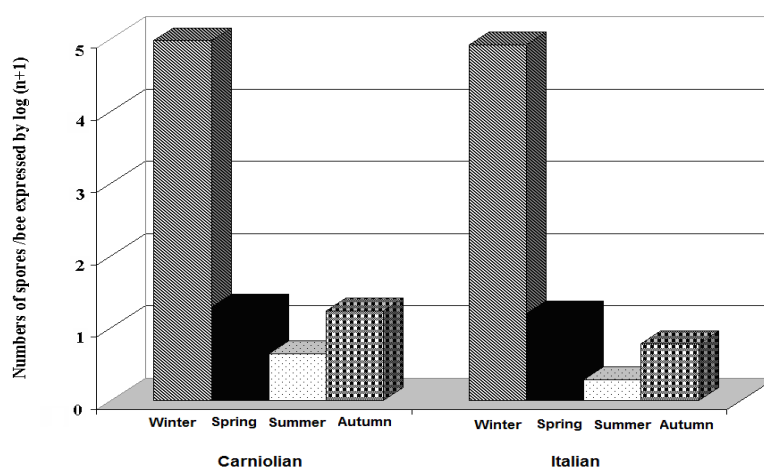


Fig. 2: Effect of *Nosema apis* infection levels on nurse bees for two honeybee hybrids during seasons of year 2011.

Table 2: Effect of *Nosema apis* infection levels in nurse bees for two honeybee hybrids during seasons of year 2011.

Season	Carniolian	Italian	F 0.05	LSD
Winter	0.94x10 <sup>6</sup>	0.82x10 <sup>6</sup>	181.5***	22669.57
Spring	193.68	157.33	647.47***	7.16
Summer	44.21	19.33	18.21*	3.58
Autumn	172.64	60.19	471.57***	4.53
F 0.05	269.64***	202.67***	-	-
LSD	9417.22	9414.22	-	-

The highest number of *Nosema* spores/bee was Carniolian bee race followed by Italian bee race.

**Carniolian bee race:** The mean number of *Nosema* spores/bee were 0.94x10<sup>6</sup>, 193.68, 172.64 and 44.21 spores/bee during winter, spring, autumn and summer respectively.

**Italian bee race:** The mean number of *Nosema* spores/bee were 0.82x10<sup>6</sup>, 157.33, 60.19 and 19.33 spores/bee during winter, spring, autumn and summer respectively.

The highest number of *Nosema* spores/bee by colony was in the winter however the lowest number of *Nosema* spores/bee there in the summer in the both two hybrids.

The number of spores/bee for each colony of the different race increased in winter and spring. It is worth nothing the summer gave the lowest number of spores.

It has been proved that the winter and spring more investigation than autumn and summer.

The statistical analysis showed that the difference of the mean number of spores/bee per colony under the different two hybrids (Carniolian and Italian) and during different seasons were significant the L.S.D, values were 22669.27, 7.16, 3.38 and 4.53 spores/ bee per colony respectively.

These result agreed with that obtained by Lotfi *et al.* (2009) determined the prevalence of *Nosema apis* in the three different seasons of spring, summer and fall in the year 2008. The infection of the honey bee colonies was of its highest level in the spring (59.5%), however the amount was considered to be low in the fall (0%) and in the summer (3.33%).and the highest level of humidity in the spring bring about *Nosema* spreads. due to the lack of humidity in the summer and fall, in these seasons the incidence of *Nosema* was observed in very lower rates. Hartwig and Topolska (1995) collected samples on 3 different dates (in January, February and March) and examined for the presence of *Nosema apis* he found that Samples from the end of the over wintering period (i.e. in February or March) contained these pathogens.

Malone *et al.* (1995). Found no significant differences among the 3 stocks of bees in the degree of this reduction in longevity. However, dark and Carniolan bees survived better in cages than Italian bees, whether dosed or not. There were significant differences among the 3 stocks in the mean numbers of spore loads, Carniolan bees the lowest, and dark bees carrying an intermediate number of spores. Thus, Carniolan bees from Australia may support a slower rate of *N. apis* proliferation and thus have lighter infections than New Zealand dark or Italian bees receiving similar doses of spores.

## 2-Histopathological studies on infection mid-gut

This disease is initiated by ingesting the highly refractile, 2x5 oval spores, which pass quickly into the midgut or ventriculus by the proventriculus. After they enter the ventriculus, they extrude their hollow polar filament and inject the germ into the epithelial cell (Morgenthaler, 1963). The parasites develop and multiply within the cytoplasm of the of the ventriculus cells. After 3 to 7 days sporonts appear in the lumen of the gut, millions of spores are shed into the digestive tract and eliminated in the faeces. Fecal contamination may be a source of infection (Moeller, 1978).

Ventriculus: To determine histopathological change produced by the *Nosema* parasite in the ventriculus, sections in healthy and infected ventriculi were examined. It was noticed that, healthy ventriculi were deeply stained and their wall consisted of single layer of columnar epithelial cells with microvilli forming a striated border, Lined with an inner circular muscles and an outer longitudinal one (Pho.1a and 1b). This striated border consisted of a fine parallel hair arising from the free epithelial cell surface and extended into ventricullus lumen. In the lumen of the vintriculus, the pritrophic membrane is concentrically arrange and extended along its entire length.

The structure of the normal honeybees ventriculus has been described by many authors (Snodgrass, 1910; White, 1919; Day and Waterhouse, 1953).

The epithelial cells of infected ventriculi were faintly stained, lost their definition and exhibited signs of lysis (Pho.3a). The cytoplasm of infected cells was largely replaced by spores. However a number of spores may leave the epithelial cells and fill the ventriculus lumen (Pho.4a, 4b and 4c). So, infection may lead to the disintegration of epithelial cell wall, striated border (Pho.2b and 2c), fragmentation of peritrophic membranes (Pho.2a). Also, destruction of the muscle layer and basement membrane was recorded (Pho.3b and 3c). Generally, the shape and arrangement of epithelial cells were changed due to the presence of spores. They tented to the retain an elongated pyriform and small cap-like shape filled by spores (Pho2b). In addition, extensive vacuolation of the cytoplasm and displacement of the nuclei were also reported (These changes may explain the increase in the ventricular epithelium thickness (Pho.2b and 2c) The modifications described in the ventriculi of infected bees, in present study were found to be similar to those reported on the same subject (Herting, 1923; Kovatschev, and Schabanov; 1972; Guzeva and Grobov, 1975; Hryniewiecka-Szyfter and Banaszak,1975; El-Shemy,1986; Duca *et al.*, 1987; Liu, 1990; Abdel-Rahman *et al.*, 2005). From the result obtained here it could be concluded that *Nosema* infected in honeybees had shown to cause atrophy in the ventricular epithelial cells, which led to gland atrophy and starvation. These events contributed to the remarked decrease in the body weight eventually early death because the digestive function of infected midgut was impaired. Therefore, the ventriculus tissue was unable to meet the bees nutritional needs. Similarly, Bailey and Ball (1991) reported a rapid decrease of dry weight in infected than uninfected bees as a result of the serious damage of infection. Nevertheless, Hassanein (1952, b) recorded an increase in the mean weight of infected bees than that of healthy one.

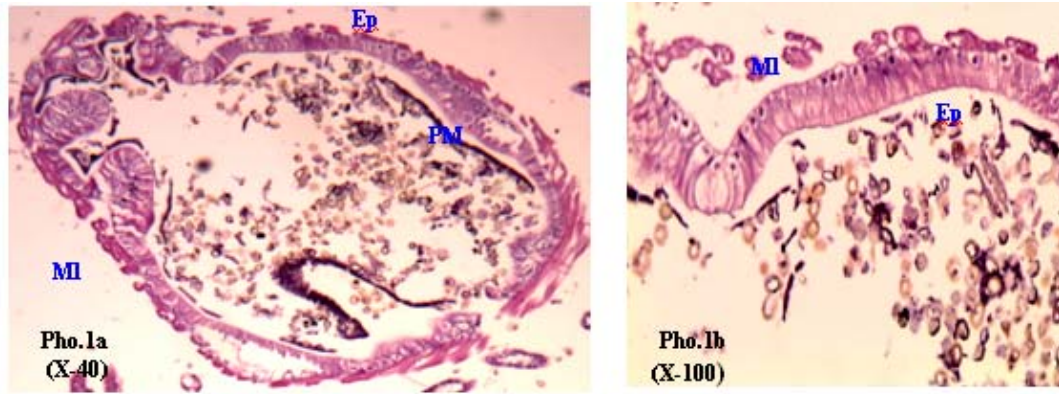
Generally, nosemosis causes heavy to the beekeeping industry (Bailey, 1955a&b; Doull, 1961; Moeller, 1962; L'Arrive,1966; Oertel,1967; Mussen *et al.*, 1975; Morse,1978), by shorten the bee's life spin (Farrar,1942; Jeffree and Allen, 1956; El-Shemy,1986; Wenning,2002b).

## REFERENCES

- Abdel-Rahman M.B, Mahmoud E.E, Manal F. E and Ghada. S.M.R (2005): Studies on some mites and protozoan parasites associated with honeybees in Egypt. Ph.D. Thesis, Fac. Sci., Cairo Univ., Egypt 127pp.
- Bailey, L. (1955a): The infection of the ventriculus of the adult honeybee by *Nosema apis* Zander. Parasitology. 45: 86-94.
- Bailey, L. and Ball, B.V. (1991): Honeybee Pathology. 2<sup>nd</sup> ed. Academic Press, San Diego, CA., 139 PP.
- Cantwell, G.E. (1970): Standard methods for counting *Nosema* spores. Am. Bee J., 110 (6): 222-223.
- Day, M.F. and Waterhouse, D.F. (1953): Function of the alimentary system. In Roeder, Insect Physiology, Chap. 11: 298-310.
- Doull, K.M. (1961): A theory of the causes of development of epizootics of *Nosema apis* disease of the honeybee. J. Insect Pathol. 3:297-309.
- Duca, C.; Papay, Z.; Miclea, M. and Muresan, E. (1987): Study of some histochemical indices in the midgut epithelium of *Apis mellifera carpatica* during infection by *Nosema apis*. Buletinul- Institutului Agonomic-Cluj-Napoca-Zootehnie-si Medicina Veterinariae, 41:41-46.
- Dyess, E.G. and Wilson, C.A. (1978): A study of the seasonal variation of *Nosema apis* Zander of honeybees in Mississippi. Am. Bee J., 118 (1):33-35.
- El-Shemy, A.A.M. (1986): The relationship between the honeybee, *Apis mellifera* L. and the sporozoan parasite, *Nosema apis* Z. Ph.D. Thesis, University of College Cardiff, Wales, U.K., 164 p.
- Farrar, C.L. (1942): *Nosema* disease contributes to winter losses and queen supersedure. Glean Bee Cult. 70 (11): 660-661.
- Graaf, D.C.de; Raes, H; Sabbe, G.; Rycke, P.H. and Jacobs, F.J. (1994): Early development of *Nosema apis* (Microspora: Nosematidae) in the midgut epithelium of the honeybee (*Apis mellifera*). J. Invert. Pathol. 63 (1): 74-81.
- Guzeva, L.N and Grobov, O.F. (1975): Interaction between *Nosema apis* and some of the bacteria in honeybees. Byulleten Vsesoyuznogo Ordena Lennina Instituta Experimental noi Veterinariii, 21:65-66.
- Hartwig, A. and Topolska, G. (1995): Value of standard testing of winter hive debris. Pszczelnicze Zeszyty Naukowe. 39 (1): 71-77.
- Hassanein, M.H. (1952a): Studies on the fluctuation in the percentage of the worker bees of the colony infected with *Nosema apis* throughout the course of the year. The Scottish Beekeeper. 28 (7): 142-143.
- Hassanein, M.H. (1952b): The effects of infection with *Nosema apis* on the pharyngeal salivary glands of the worker honeybee. Proc. Poy. Ent. Soc. London, 27:22-27.
- Herting, M. (1923): The normal and pathological histology of the ventriculus of the honeybee, with special reference to infection with *Nosema apis*. J. Parasitol. 9 (3): 109-140.
- Hryniewiecka-Szyfter, Z. and Banaszak, J. (1975): Histological investigation of the midgut of bees infected with the spores of *Nosema apis* Zander by individual and mass feeding Bull Soc. Amis des Sci. et Letters de Poznan D.(Sciences Biologiques), No.15: 83-86.
- Jeffree, E.P. and Allen, N.D. (1956): The influence of colony size and of *Nosema* disease on the rate of population loss in honeybee colonies in winter. J. Econ. Entomol. 49 (6): 831-834.

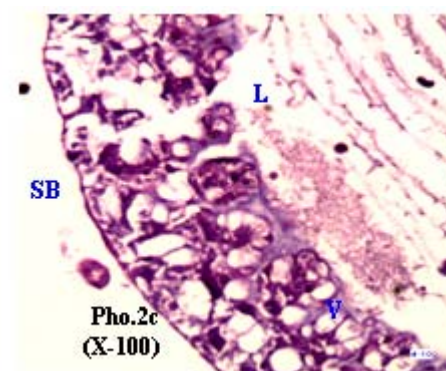
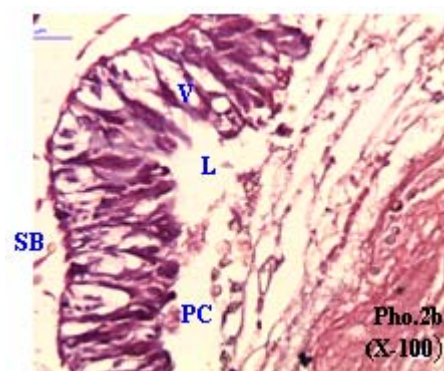
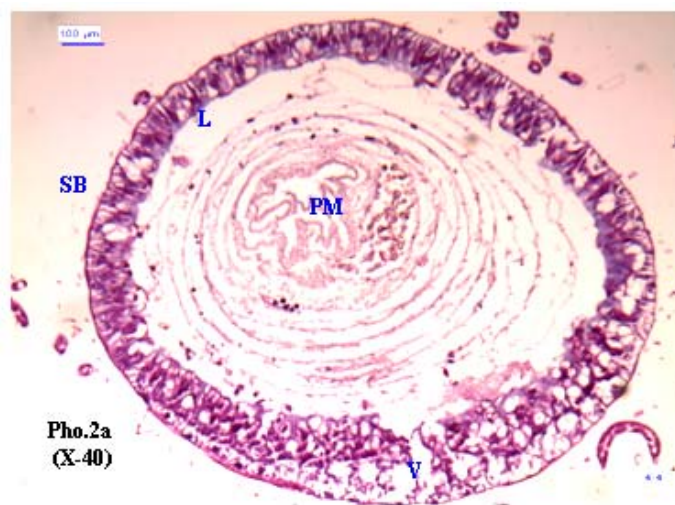
- Kovatschev, K. and Schabanov, M. (1972): Histological change in the midgut wall of worker bees infected with *Nosema apis*. Archiv. Exp. Veterinarmedizin, Bulgaria., 26 (3): 455-460.
- L' Arrivee, G.C.M. (1966): Effects of *Nosema* disease on honey yield. Can. Agric., 11 (3): 24-25.
- Liu, T.P. (1990): The release of *Nosema apis* spores from epithelium of the honeybee midgut. J. Apic. Res., 29 (4): 221-229.
- Lotfi, A.; Jamshidi, R.; Shahryar, H. A. and Yousefkhani, M. (2009): The prevalence of *Nosemosis* in honey bee colonies in Arasbaran region (northwestern Iran). American-Eurasian J. Agric. Environmental Sci., 5 (2): 255-257.
- Malone, L. A.; Giacon, H. A. and Newton, M. R. (1995): Comparison of the responses of some New Zealand and Australian honey bees (*Apis mellifera* L.) to *Nosema apis* Z. Apidologie., 26 (6): 495-502.
- Moeller, F.E. (1962): *Nosema* disease control in backage bees. Am. Bee J., 102 (10): 390-392.
- Moeller, F.E. (1978): *Nosema* disease, its control in honeybee colonies. Tech. Bull.Dept.of Agric., USA, No. 1569, 16 pp.
- Morgenthaler , O. (1963): The germinationof *Nosema* spores Imker., 15 (4): 102-104.
- Morse, R.A (1978): Honeybee pests, predators and disease. Cornell Univ. Press, Ithaca, New York, 430 pp.
- Muresan, E.;Duca, D. and Papay, Z. (1975): The study of some histochemical indices of the midgut,healthy and infected with *Nosema apis*, of the *Apis mellifica* carpatica bee. In: Pro. XXV<sup>th</sup> Int. Api. Congr., pp. 384-385.
- Mussen, E.C.; Furgala, B. and Hyser, R.A. (1975): Enzootic levels of *Nosema* disease in the contimenatal United States. Am. Bee J., 115 (2): 48-50.
- Oertel, E. (1967): Colony disturbance and *Nosema* disease. J. Apic. Res., 6 (2): 119-120.
- Snodgrass, R.E. (1910): The Anatomy of the Honeybee. 162 pp., U.S Dep. Agric., Bur. Ent. Tech. Ser., No. 18.
- Thomas, G.; Tracey, B.; Francesca, G. and Michael, H. (2009): *Nosema ceranae* infects honey bees (*Apis mellifera*) and contaminates honey in Australia. Apidologie., 40:117-123.
- Wenning, C. J. (2002b): Reduced chemical beekeeping 111. Amer. Bee. J., 142 (3):205-209.
- White, G.F. (1919): *Nosema* Disease. USDA Bull. no. 780, Washington, D. C., 59 PP.
- Yanping Chen, Jay D. Evans, I. Bart Smith, Jeffery S. Pettis. (2008): *Nosema ceranae* is a long-present and wide-spread microsporidian infection of the European honey bee (*Apis mellifera*) in the United State. J. Invertebr. Pthol., 97:184-188.





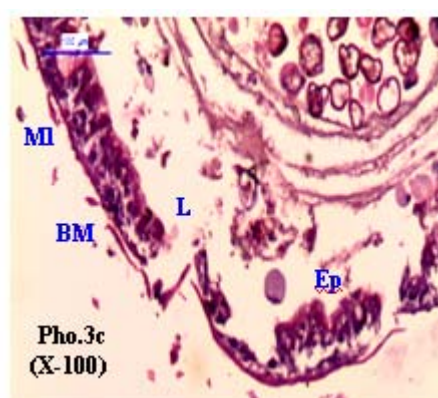
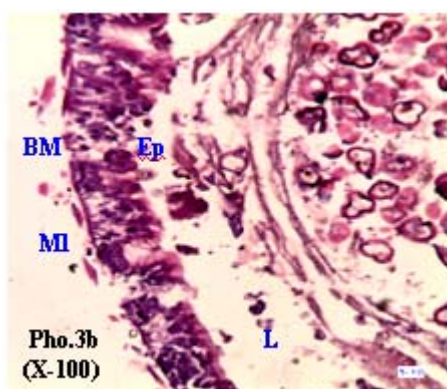
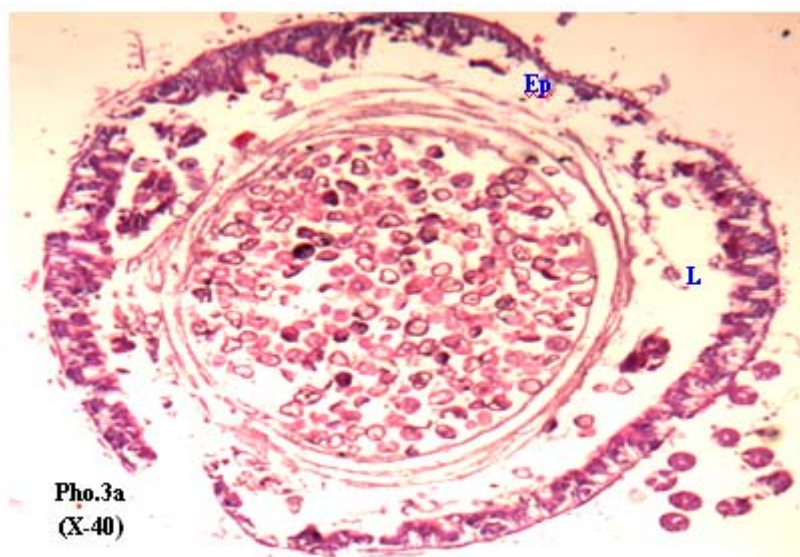
(Photo1a). Transverse section through the ventriculus of the healthy worker bee. Ep, epithelium; PM, peritrophic membranes. (X-40)

(Photo1b). Enlarged part show. Ep, epithelium; ML, muscle layers. (X-100)



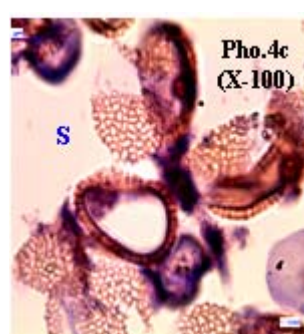
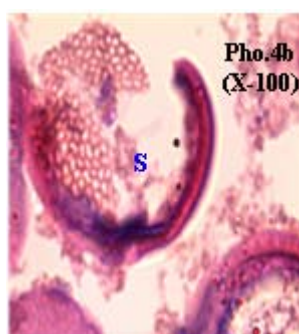
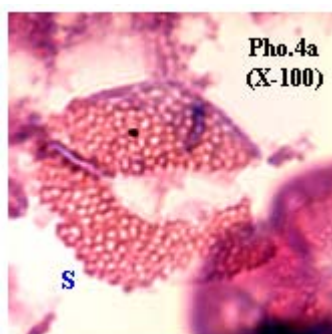
(Photo2a). Transverse section through the ventriculus epithelium of *Nosema* infected worker honeybee. PM, peritrophic membranes; L, lumen; V, vacuole; SB, striated border. (X-40)

(Photo2b&Photo2c). Enlarged part show. L, lumen; V, vacuole; PC, pyriform cells with spores, Note disintegration of cell wall SB, striated border. (X-100).



(Photo3a). Transverse section through the ventriculus of heavy *Nosema* infected worker bee. Ep, epithelium. L, lumen. (X-40)

(Photo3b & Photo3c). Enlarged part show. Ep, epithelium. L, lumen; MI, muscle layers; BM, basement membrane. Note the lysis of epithelial cells, disintegration of muscle layers and basement membrane. (X-100)



(Photo 4). *Nosema* spores in lumen of the ventriculus of *Nosema* infected worker honeybees. S, *Nosema* spores. (X-1000)

## ARABIC SUMMARY

العلاقة بين مستوى الإصابة بطفيل النوزيما والخلايا الظلانية للمعي المتوسط لنحل العسل بمحافظة قنا

محمد زكي يوسف علي<sup>1</sup> – كارم محمد مهني عبد العال<sup>2</sup> – هدي سعدي محمد<sup>1</sup>

رشا عبد الله سيف النصر<sup>1</sup>

1- قسم علم الحيوان – كلية العلوم بقنا – جامعة جنوب الوادي

2- قسم وقاية النبات – كلية الزراعة بقنا – جامعة جنوب الوادي

كان الهدف من البحث الذي اجري في منحل كليه الزراعة بجامعة جنوب الوادي بقنا خلال الفترة 2011 لسالتين من النحل الايطالي والكرنولي دراسة العلاقة بين مستوى الإصابة بطفيل النوزيما والخلايا المبطنة لجدار المعى المتوسط .  
أوضحت النتائج ان هناك فروق معنوية عالية بين متوسط اعداد الجراثيم حيث كانت اعداد الجراثيم في السلالة الكرنولي اعلي من السلالة الايطالي (20467.26-11402.03) خلال فتره الدراسة بالتتابع .  
كما اظهرت القطاعات الهستولوجية انه في حاله الإصابة الشديدة حدث تحلل للخلايا المبطنة لجدار المعى المتوسط .  
اما في حاله الإصابة المتوسطة ظهرت الخلايا بالحالة شبه متحللة مقارنة بالخلايا السليمة .  
ويوصي البحث بتكثيف برامج العلاج والاهتمام خلال اشهر شدة الاصابه (يناير،فبراير،مارس) بمحافظة الجنوب.