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# SURVEILLANCE OF *TRYPANOSOMA SPP* OF RODENTS AND STUDIES IN THEIR TRANSMISSION PROBABILITY BY FLEAS IN SOME RURAL EGYPTIAN AREAS

By

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**Abstract**

A new public health problem arises from animal trypanosomes that afflict human by a disease called atypical human trypanosomiasis. Although humans have an innate protection against most *Trypanosoma* species, nineteen cases of atypical human trypanosomiasis caused by the animal trypanosome as *T. b. brucei* , *T. vivax*, *T. congolense* , *T. evansi* and *T. lewisi* have been recorded. Some of theserecorded cases were transient, six required trypanocidal treatments however two patients died. Rodent trypanosome, *T.lewisi* is transmitted via ingestion of fleas or their feces containing the infective stage, the metacyclic trypomastigote. Because of the high densities of various species of rodents and their distribution all over the country especially in rural areas, the present work aimed to evaluate the trypanosomiasis among rodents collected from November to March 2016 and study transmission probability by their fleas in some rural areas in Abu Alnomros Center, Giza. The overall trypanosomiasis prevalence among the different rodent species was (21 rats) 24.7%. All the infected rats belonged to *Rattus r. spp* where the prevalence of infection with *Trypanosoma lewisi* among that species was very high 51.2% while none of rats belonged to *Rattus norvegicus* were infected. That may be attributed to the solid immunity gained by the *R. norvegicus* where most of the collected norvegicus were aged and weighed more than 200 grams. There was an inverse significant correlation between the densities of parasites and the weights of the hosts. The rat which recorded the highest parasite density (60,000 parasites/ microliter) was a female *Rattus r.* captured indoor (inside house). As to sex of *Rattus rattus spp* no significant difference was found between males and females in trypanosomiasis. Also there was no significant correlation between the densities of parasites and the number of white blood cells among *Rattus rattus spp*. All positive rats were collected indoors (from houses) and all the rats which were captured from outdoors (farms) were negative for *T. lewisi*. The difference between infections with trypanosomiasis among rats inhabited the houses and that found in farms was highly significant.

Only two species of fleas were found on rats, *Xenopsylla cheopis* and *Leptopsylla segnis.* The oriental fleas, *X. cheopis*, were found mainly on *R. norvegicus* where 57.5% of *R. norvegicus* were positive for *X. cheopis* while only one rat was positive for *L. segnis*. On other hand the rat fleas, *L.segnis*, were found mainly on *Rattus rattus* spp where 39% of these rats were positive for *L. segnis*. The present work revealed a significant correlation between the infection with *T. lewisi* and the presence of *L. segnis* on the rats however that correlation regarding *X. cheopis* was not significant.

## Introduction

Trypanosomes are protozoan parasites widely distributed, infecting humans, wild and domestic animals. The typical pathogenic human trypanosomes cause sleeping sickness disease, or human African trypanosomiasis (Hoare, 1972), and the Latin American Chagas disease (Rassi et al, 2010). Human African trypanosomiasis is a fatal disease found in sub-Saharan Africa and transmitted by tsetse flies. The disease is caused by two subspecies of trypanosomes; *T. brucei gambiense* which is responsible for the chronic form or *Trypanosoma b.*

*rhodesiense* that causes the acute form. Chagas disease is caused by *Trypanosoma cruzi*, and transmitted mainly by triatomine bugs (Maraghi, 1995). The disease is endemic in Latin America where most cases are chronic and asymptomatic (Rassi *et al*, 2010).

On other hand, most of trypanosomes were thought to be infective only to animals, such as *T. congolense*, *Trypanosoma b. brucei,* and *T. vivax*, that are responsible for "nagana" or the complex animal trypanosomiasis in Africa. *T. evansi* is the causative agent of a disease called "surra" that is widely distributed among wild and domestic animals. Surra is found in Asia, Africa, Europe, and even South America (Gutierrez, 2010).

Man has an innate protection against most *Trypanosoma* sp. (Vanhamme 2003). However, nineteen cases of atypical human trypanosomiasis caused by *T. b. brucei*, *T. vivax* (Hoare, 1972), *T. congolense* (Truc *et al,* 1998), *T. evansi* (Joshi *et al,* 2005) and *T. lewisi* (Doke *et al,* 2011; Verma *et al,* 2011), which were previously non-infective to humans, have been reported. In recent years, *T. lewis* and *T. evansi* have emerged as potential pathogens for humans. Most of the recorded cases were transient, six cases required trypanocidal treatments while two of them died (Doke *et al,* 2011). Among fifteen humans cases reported in the period between 1974 and 2010, nine of them have been recorded since 2003. Some cases were identified by microscopic observation of trypanosomes while others by using molecular tools (Truc *et al,* 2013). Haridy *et al*. (2011) in Egypt reported camels naturally infected with *T. evansi* and the first Egyptian human case of zoonotic trypanosomiasis *evansi*.

In a trial for detecting the possible source of infection with atypical trypanosomiasis in a sick Thai infant, blood samples of 276 rodents were analyzed for identifying ITS1 sequence of trypanosomes. The result revealed that there was 96.4% similarity between ITS1 sequence of trypanosome obtained from *R. tanezumi* and that from the blood of the *T. lewisi*-infected Thai infant (Sarataphan *et al,* 2007; Desquesnes *et al,* 2011). *T. lewisi* possess two antigenic variants the first one represents the initial reproducing population while the second represent the non-reproducing population. The reproduction rate is inhabited by ablastin (WHO, 1970) while the late population is cleared by antibody dependent cytotoxicity. During Infection of rat with *T. lewisi* the parasitaemia naturally resolves within 30 days providing solid immunity against reinfection (Maia da Silva, 2010). Inability of *T. lewisi* to infect a wide range of mammalian species may be due to the activation of complement through the alternative pathway, agglutinins and opsonins where the parasites infecting non-natural hosts are cleared in a very short time compared to their natural hosts (Jarvinen *et al,* 1976; Desquesnes *et al,* 2011).

The rodent species found in Egypt Governorates were Norway rat, *Rattus norvegicus*, white-bellied rat, *R. rattus frugivorous*, the grey-bellied rat, *R. r. alexandrinus*, house mouse, *Mus musculus* and finally the spiny mouse *Acomys cahirinus*. The flea species attacking rodents were the common species or the rat flea, *Xenopsylla cheopis* then the mouse flea, *Leptopsylla segnis*. Also other fleas as dog flea, *Ctenocephalides canis* and the sticktight flea *Echidnophaga gallinacean* were recorded. Highest flea indices were detected in Ismailia and Matrouh Governorates but El-Fayoum and North Sinai Governorates recorded the lowest ones. *R. norvegicus* was the highest manifested species with fleas because they live in places easy to dig barrows and also are suitable condition for fleas breeding. The lowest manifestation by fleas was recorded on *Mus musculus* and *Acomys cahirinus. X. cheopis* was the species of the highest frequent distribution on all domestic rodent species however the stick-tight flea, *Echidnophaga gallinacea* was the species which recorded the lowest distribution where they found only in Dakahlia and Ismailia (Mikhail *et al,* 2011).

The present work aimed to evaluate the trypanosomiasis among rodents collected from November to March 2016 and to study transmission probability by their fleas in some rural areas in Abu Alnomros Center, Giza Governorate.

## Subjects, Material and Methods

Well baited wire box traps were distributed at sunset in some selected residential rural houses in Abu Al-nomros Center, Giza Governorate. Distributed traps were collected next morning, enclosed with separate white bags and transported to laboratory.

Capturing and transporting of rodent animals throughout the present investigation were done (Rifaat *et al,* 1969).The collected animals (85 rats) were identified then anaesthetized with diethyl ether. Fleas on each animal were collected in white sheet by using a stiff hard brush (WHO, 1970). Fleas were preserved in 70% ethanol in separate labeled tube. Classification and Identification of fleas were done according to the key given by Hoogstraal (1956).

Blood samples were taken from hearts of the anaesthetized rats. Thin and thick blood films were prepared and stained with Giemsa stain to demonstrate the *Trypanosoma spp*. (Gracia, 2001).

The collected data were tabulated and statistically analyzed by PC using the Epi Info and SPSS for windows software packages. The 0.05 cut-off value was used as a criterion for statistical significance and all statistical tests were interpreted in a two-tailed fashion (Lehmann, 1975, Altman, 1992).

## Results

The results were shown in tables (1 to 11), figures (1 to 10), and photos (1 to 6).

## Discussion

In the present study the overall trypanosomiasis prevalence among the different rodent species was (21 rats) 24.7%. All the infected rats belonged to *Rattus rattus spp* where the prevalence of infection with *Trypanosoma lewisi* among that species was very high 51.2%. Regardless the rodent species the overall prevalence in the present study is close to that (21.7%) recorded in Belo Horizonte, State of Minas Gerais, Brazil (Linardi *et al,* 2002) also in Venezuela which recorded 21.3% (Herrera *et al,* 1997) and in Italy 20% (De Carnieri *et al,* 1964). The highest recorded prevalence was found in India 82.3% (Laha *et al,* 1997). Other prevalences of rodent trypanosomiasis in different areas all over the world were 8.9% in Nigeria (Ugbomoiko, 1997), 13.2% in both in Egypt (Abdel-Aal *et al,* 1997) and the USA (Eyles, 1952).

Regarding rodent species the prevalence of trypanosomiasis among *Rattus rattus spp* was 51.2% while no positive Norway rats (*Rattus norvegicus*) were recorded in the present work. That may be due to most of the collected *Rattus norvegicus* 95.4% recorded weights equal or more than 100 grams and 86.4% weighed 200 grams or more. The high weights of the collected Norway rats reflect their ages and their corresponding immunity against trypanosomiasis.

The multiplication of *T. lewisi* in rodents is well known (Albright *et al,* 1991). After a period of rapid multiplication of trypanosomes (10 days), they stop growing and their numbers stabilize for several weeks, then the parasites disappear suddenly from the blood so the rat develops a solid immunity against *Trypanosoma lewisi* reinfection. On other hand a trick made by *T. lewisi* minimizes the effects of the host immune response known as mimicry. Firstly the parasites coat their surfaces with a host protein layer, ablastin, identified as IgE that forbids the trypanosomes from dividing or multiplying. After several weeks, IgM antibodies are produced by the host and recognize this complex on the parasite's surface and then the complement system is activated resulting in rapid lysis of the parasites in the blood. So, the blood smears of adult rats show low densities or fewer trypanosomes than those of younger or immature animals. Since Norway rats attain sexual maturity at 75 days and only 5% of the population survives 12 months (Davis, 1948), the rats most likely to

be infected before or during their first mating. So, it is recommended to use hemoculture rather than stained blood films to show infections in many rats, mainly adults that would be considered negative by blood films (Linardi *et al,* 2002). Although no trypanosomiasis was found among *R. norvegicus* in the present study, Chaisiri (2015) mentioned that the rats belonged to that species were identified as reservoirs for most of zoonotic helminthes.

Regarding the sex of *R. rattus spp* no statistically significant difference was found between males and females in trypanosomiasis. That result agreed with studies of Franjola *et al.* (1995) and Ugbomoiko, (1997) and disagreed with Linardi *et al.* (2002) who found that the infection among males of *R norvegicus* was significantly higher than that of females. He mentioned that the higher prevalence of infection among males could be attributed to behavioral and ecological factors. The higher prevalence could be attributed to ecological and behavioral factors. For examples male rats have larger home ranges as they show territorial behavior and in turn they are significantly more infected by *X. cheopis* than females. So their chances of being infected with trypanosomiasis increased (Linardi *et al,* 1985a).

There was no significant correlation between the densities of parasites and the number of white blood cells among *R. rattus spp*. However, there was an inverse statistically significant correlation between densities of parasites and the weights of the hosts. The rat which recorded highest parasite density (60,000 parasites/ microliter) was a female *R. rattus* captured indoor. Its white blood cells WBCs count was 20,000/ microliter and its weight was 141 grams while the rat with lowest density (260parasites/ microliter) was a female weighed 163 grams, captured indoor and its WBCs count was 6500/microliter. The inverse correlation between the densities of the parasites and the weights of rats can be explained by the multiplication profile of *T. lewisi* which starts exponential multiplication then the parasites number stabilizes and is cleared by immune response. The first exposure to *T. lewisi* provides the host long solid immunity (Albright et al, 1991). So in the present study most of infected rats which recorded the high densities of parasite were of low weights or young ages. A similar study on the relation between the rates of *T. lewisi* infection and total body length of *R. norvegicus* revealed that there was a gradual increase of infection rates with total body length in animals measuring (60-170 mm) and decreased thereafter. The highest rates were detected among young animals measuring (141-170 mm) and immatures rats measuring (111-140 mm). Significant differences were detected between the infection rates of length measuring (141-170 mm) and that of rats measuring (171-200 mm) also between the infection rate of the latter group and that of rats measuring (201-230 mm) (Linardi *et al,* 2002).

Concerning the place of rodents capturing it was found that all positive rats were collected indoors (from houses) and all the rats captured from outdoors (farms) were negative for *T. lewisi*. The difference between infections with trypanosomiasis among rats inhabited the houses and that found in farms was highly significant. This result coincides with Pumhom who recorded a high prevalence of *T. lewisi* infection among rodents living near human settlement and in areas having high cover of built-up habitat (Pumhom *et al,* 2015). The results suggested strongly that the wild rodents act as reservoirs and serious source of atypical human trypanosomiasis caused by animal trypanosomes.

In the present study (November to March 2016) the estimated prevalence may not reflect the all over rate. Linardi (2002) recorded *T. lewisi* infection among *R. norvigicus* throughout the year except during February and March. Linardi (1985a,b) mentioned that the highest prevalences were recorded

in the warm and rainy season especially in October and November.

Only two species of fleas were found on the collected rats, *Xenopsylla cheopis* and *Leptopsylla segnis.* The oriental fleas, *X. cheopis*, were found mainly on *R. norvegicus* where 57.5% of *R. norvegicus* were positive for *X. cheopis* while only one rat was positive for *Leptopsylla segnis*. On other hand the rat fleas, *L. segnis*, were found mainly on *Rattus rattus spp* where 39% of these rats were positive for *L. segnis*. In the spring seasons of 2009-2010 a survullence of domestic rodents and their fleas revealed that *X. cheopis* were the fleas of the highest frequent distribution on all domestic rodents (Mikhail *et al,* 2011).

The present work revealed a significant correlation between the presence of *Leptopsylla segnis* on the rats and the infection with *T. lewisi* however that correlation regarding *Xenopsylla cheopis* was not significant. Linardi (1985a,b) revealed that the high level of infestation by *X. cheopis* coincide with high infection rate with *T. lewisi* .

## Recommendation

Although few number of atypical human trypanosomiasis appeared recently all over the world, the high densities of the reservoir hosts (rodents) and flea vectors in our communities threaten people especially the classes of low immunity with infection by *T. lewisi* .So regular survey for rodents all over the country should be done. A plan for controlling fleas and rodents should be achieved. On research level molecular studies on the two species; *Xenopsylla cheopis* and *Leptopsylla segnis* should be done for detecting the vector which is responsible for transmission of *T. lewisi*.

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Table 1: Prevalence of infection with *T. lewisi* among rodents collected from Abu Alnomros Center. $X^2$ =29.9, P < 0.01

| Rodent species | | Trypanosomiasis *lewisi* | | Total |
|---|---|---|---|---|
| | | Negative | positive | |
| *R. norvegicus* | No. of rodents | 44 | 0 | 44 |
| | % | 100.0% | 0.0% | 100.0% |
| *R. rattus* | No. of rodents | 20 | 21 | 41 |
| | % | 48.8% | 51.2% | 100.0% |
| Total | No. of rodents | 64 | 21 | 85 |
| | % | 75.3% | 24.7% | 100.0% |

Table 2: Distribution of different rodents collected from Abu Alnomros Center, according to capturing place. $X^2$ =27.6, P < 0.01

| Species of rodents | | Place of rodent capturing | | Total |
|---|---|---|---|---|
| | | Indoors | Outdoors | |
| *R. norvegicus* | No. of rodents | 22 | 22 | 44 |
| | % | 50.0% | 50.0% | 100.0% |
| *R. rattus* | No. of rodents | 41 | 0 | 41 |
| | % | 100.0% | 0.0% | 100.0% |
| Total | No. of rodents | 63 | 22 | 85 |
| | % | 74.1% | 25.9% | 100.0% |

Table 3: Rodents collected from Abu Alnomros Center, according to sex and infection with trypanosomiasis. $X^2$ =0.63, 0.04, P > 0.05

| Rodent Species | | | *Trypanosoma lewis* | | Total |
|---|---|---|---|---|---|
| | | | Negative | Positive | |
| *R. norvegicus* | Male | No. of rodents | 25 | 0 | 25 |
| | | % | 100.0% | 0 | 100.0% |
| | Female | No. of rodents | 19 | 0 | 19 |
| | | % | 100.0% | 0 | 100.0% |
| | Total | No. of rodents | 44 | 0 | 44 |
| | | % | 100.0% | 0 | 100.0% |
| *R. rattus* | Male | No. of rodents | 8 | 11 | 19 |
| | | % | 42.1% | 57.9% | 100.0% |
| | Female | No. of rodents | 12 | 10 | 22 |
| | | % | 54.5% | 45.5% | 100.0% |
| | Total | No. of rodents | 20 | 21 | 41 |
| | | % | 48.8% | 51.2% | 100.0% |
| Total | Male | No. of rodents | 33 | 11 | 44 |
| | | % | 75.0% | 25.0% | 100.0% |
| | Female | No. of rodents | 31 | 10 | 41 |
| | | % | 75.6% | 24.4% | 100.0% |
| | Total | No. of rodents | 64 | 21 | 85 |
| | | % | 75.3% | 24.7% | 100.0% |

Table 4: Distribution of different rodents collected from Abu Alnomros Center, according to flea spp. $X^2$ =24.8, P < 0.01

| Rodent Species | | Presence of Fleas | | | | Total |
|---|---|---|---|---|---|---|
| | | Negative | *Xenopsylla spp* | *Leptopsylla spp* | Both fleas | |
| *R. norvegicus* | No. of rodents | 20 | 23 | 0 | 1 | 44 |
| | % | 45.5% | 52.3% | 0.0% | 2.3% | 100.0% |
| *R. rattus* | No. of rodents | 20 | 5 | 6 | 10 | 41 |
| | % | 48.8% | 12.2% | 14.6% | 24.4% | 100.0% |
| Total | No. of rodents | 40 | 28 | 6 | 11 | 85 |
| | % | 47.1% | 32.9% | 7.1% | 12.9% | 100.0% |

Table 5: Correlation between *Xenopsylla spp* on rats and infection with *T. lewisi.*

| | | *Xenopsylla spp* | Trypanosomiasis |
|---|---|---|---|
| *Xenopsylla spp* | Pearson Correlation | 1 | .029 |
| | Sig. (2-tailed) | | .793 |
| | N | 85 | 85 |
| Trypanosomiasis | Pearson Correlation | .029 | 1 |
| | Sig. (2-tailed) | .793 | |
| | N | 85 | 85 |

Table 6: Correlation between *Lepytopsylla spp* on rats and infection with *T. lewisi.*

| | | Trypanosomiasis | *Lepytopsylla spp* |
|---|---|---|---|
| *Lepytopsylla spp* | Pearson Correlation | 1 | .427** |
| | Sig. (2-tailed) | | .000 |
| | N | 85 | 85 |
| Trypanosomiasis | Pearson Correlation | .427** | 1 |
| | Sig. (2-tailed) | .000 | .000 |
| | N | 85 | 85 |
| ** Spearman's rho correlation significant at 0.01 level (2-tailed). | | | |

Table 7: Correlation between weights of rats and infection with *T. lewisi.*

| | | Weights | Trypanosomiasis |
|---|---|---|---|
| Weights | Correlation Coefficient | 1.000 | -.500-** |
| | Sig. (2-tailed) | . | .000 |
| | N | 85 | 85 |
| Trypanosomiasis | Correlation Coefficient | -.500-** | 1.000 |
| | Sig. (2-tailed) | .000 | . |
| | N | 85 | 85 |
| **. Spearman's rho correlation significant at 0.01 level (2-tailed). | | | |

Table 8: Rodents collected from Abu Alnomros Center, according to weights and trypanosomiasis infection $X^2 = 23.6$, $P < 0.01$

| Weights of different species | | Negative for *T. lewisi* | | positive for *T. lewisi* | | Total | |
|---|---|---|---|---|---|---|---|
| | | No. | % | No. | % | No. | % |
| *R. norvegicus* | Less than 100 | 2 | 4.5% | | | 2 | 4.5% |
| | 100 - less than 200 | 4 | 9.1% | | | 4 | 9.1% |
| | 200 - less than 300 | 19 | 43.2% | | | 19 | 43.2% |
| | 300 - less than 400 | 17 | 38.6% | | | 17 | 38.6% |
| | 400 grams or more | 2 | 4.5% | | | 2 | 4.5% |
| | Total | 44 | 100.0% | | | 44 | 100.0% |
| *R. rattus* | Less than 100 | 7 | 35.0% | 10 | 47.6% | 17 | 41.5% |
| | 100 - less than 200 | 13 | 65.0% | 11 | 52.4% | 24 | 58.5% |
| | Total | 20 | 100.0% | 21 | 100.0% | 41 | 100.0% |
| Total | Less than 100 | 9 | 14.1% | 10 | 47.6% | 19 | 22.4% |
| | 100 - less than 200 | 17 | 26.6% | 11 | 52.4% | 28 | 32.9% |
| | 200 - less than 300 | 19 | 29.7% | 0 | 0.0% | 19 | 22.4% |
| | 300 - less than 400 | 17 | 26.6% | 0 | 0.0% | 17 | 20.0% |
| | 400 grams or more | 2 | 3.1% | 0 | 0.0% | 2 | 2.4% |
| | Total | 64 | 100.0% | 21 | 100.0% | 85 | 100.0% |

Table 9: White blood cells count/microliter & trypanosomiasis among rats from Abu Alnomros Center, $X^2 = 7.1$, $P > 0.05$

| WBCs count /microliter | | No. of parasites /microliter | | | | Total |
|---|---|---|---|---|---|---|
| | | Negative | less than 1000 | 1000-<10,000 | 10,000-60,000 | |
| Less than 5000 | No. | 15 | 2 | 9 | 0 | 26 |
| | % | 57.7% | 7.7% | 34.6% | 0.0% | 100.0% |
| 5000 - less than 10,000 | No. | 18 | 1 | 0 | 1 | 20 |
| | % | 90.0% | 5.0% | 0.0% | 5.0% | 100.0% |
| 10000-less than 20000 | No. | 16 | 3 | 0 | 2 | 21 |
| | % | 76.2% | 14.3% | 0.0% | 9.5% | 100.0% |
| more than 20000 | No. | 16 | 0 | 0 | 2 | 18 |
| | % | 88.9% | 0.0% | 0.0% | 11.1% | 100.0% |
| Total | No. | 65 | 6 | 9 | 5 | 85 |
| | % | 76.5% | 7.1% | 10.6% | 5.9% | 100.0% |

Table 10: Correlation between weights of rats and densities of *T. lewisi.*

| | | Parasite/microliter | Weight |
|---|---|---|---|
| Parasite/ Microliter | Correlation Coefficient | 1.000 | -.481-** |
| | Sig. (2-tailed) | . | .000 |
| | N | 85 | 85 |
| Weight | Correlation Coefficient | -.481-** | 1.000 |
| | Sig. (2-tailed) | .000 | . |
| | N | 85 | 85 |
| ** Spearman's rho correlation significant at 0.01 level (2-tailed). | | | |

Table 11: Different rodents collected from Abu Alnomros Center, according place and fleas spp. $X^2 = 2.8, 4.6$, $P > 0.01$

| Species | | Negative | | | | | | | | *Xenopsylla spp* | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Indoor | | 8 | | *Leptopsylla spp* | | Both fleas | | | |
| *R. norvegicus* | Outdoors | 12 | 36.4% | 14 | 63.6% | 0 | 0.0% | 0 | 0.0% | 22 | 100.0% |
| | Total | 20 | 54.5% | 9 | 40.9% | 0 | 0.0% | 1 | 4.5% | 22 | 100.0% |
| | Indoor | 20 | 45.5% | 23 | 52.3% | 0 | 0.0% | 1 | 2.3% | 44 | 100.0% |
| *R. rattus* | Total | 20 | 48.8% | 5 | 12.2% | 6 | 14.6% | 10 | 24.4% | 41 | 100.0% |
| | Indoor | 28 | 48.8% | 5 | 12.2% | 6 | 14.6% | 10 | 24.4% | 41 | 100.0% |
| Total | Outdoors | 12 | 44.4% | 19 | 30.2% | 6 | 9.5% | 10 | 15.9% | 63 | 100.0% |
| | 40 | 47.1% | 54.5% | 9 | 40.9% | 0 | 0.0% | 1 | 4.5% | 22 | 100.0% |
| Total | | | | | | | | | | | |

*Fig* **Explanation of figures**

Fig 1: *T. lewisi* among different rodents collected from Abu Alnomros Center.
Fig. 2: Rodent species collected from Abu Alnomros Center, place of rodent capturing.
Figs. 3, 4: Rodent species collected from Abu Alnomros Center, sex and trypanosomiasis infection.
Fig. 5: Rodent species collected from Abu Alnomros Center, Alnomros Center, fleas *spp.*
Fig. 6: White blood cells count / microliter and trypanosomiasis among rats in Abu Alnomros Center.
Fig. 7: White blood cells count / microliter and densities of parasites/microliter among rats in Abu Alnomros Center.
Fig. 8: Rodent species collected from Abu Alnomros Center, weights and trypanosomiasis infection.
Figs. 9, 10: Rodent species collected from Abu Alnomros Center, place of capturing and fleas *spp.*

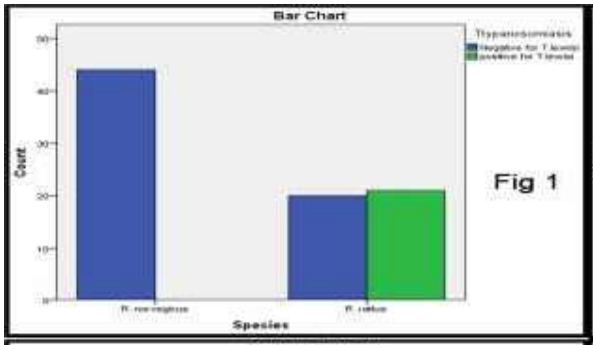


Fig 1

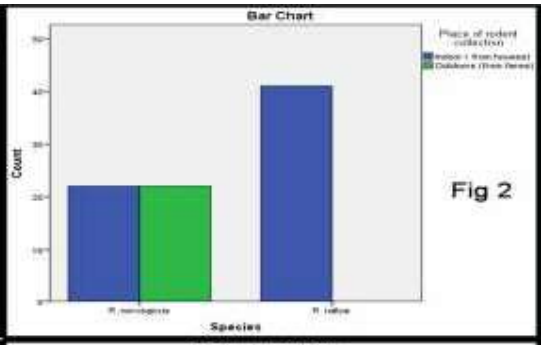


Fig 2

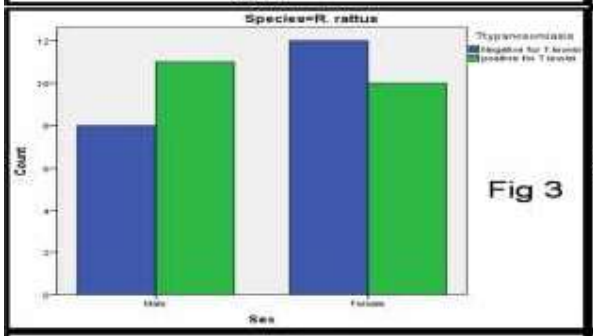


Fig 3

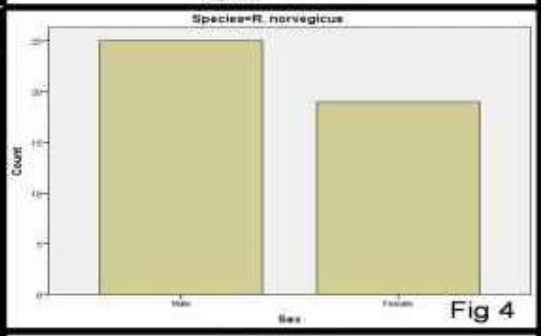


Fig 4

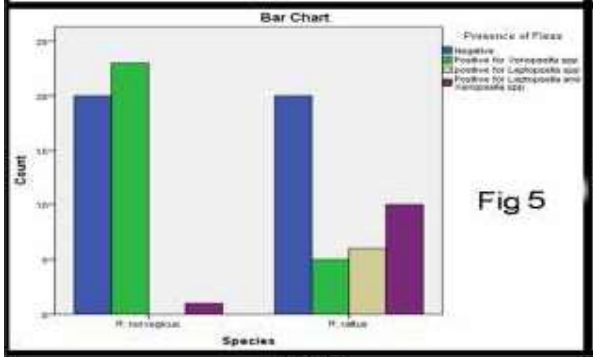


Fig 5

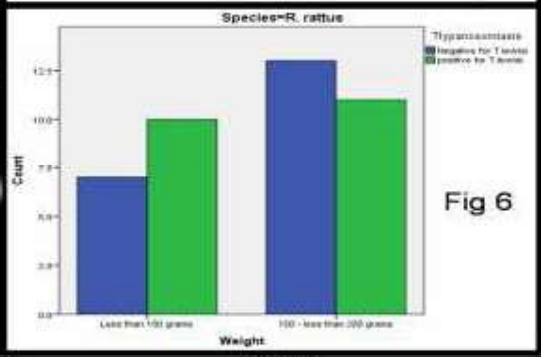


Fig 6

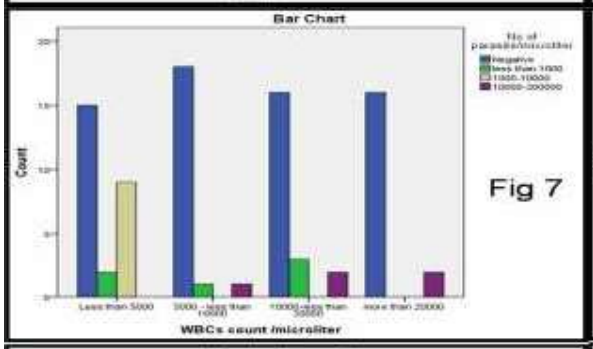


Fig 7

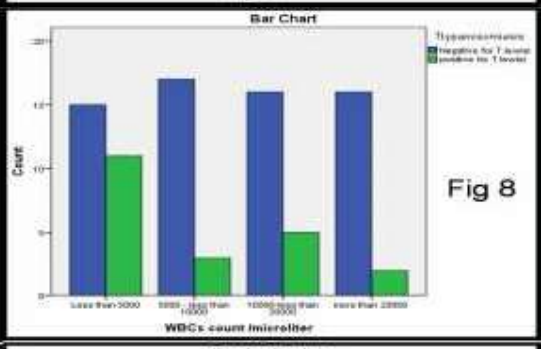


Fig 8

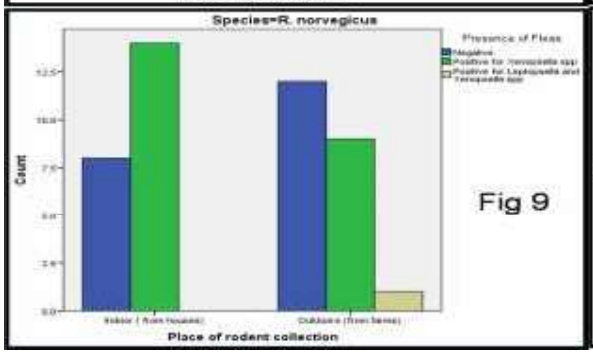


Fig 9

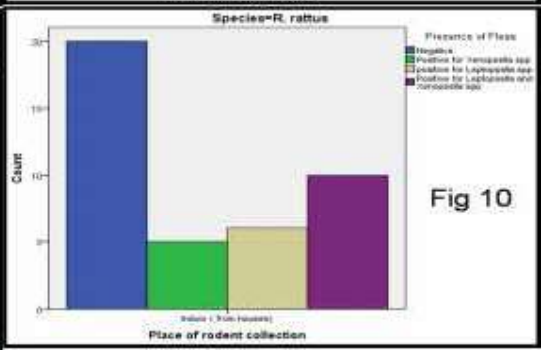


Fig 10

**Explanation of photos**
Photo 1: *Rattus norvegicus*
Photo 2: *Rattus rattus frugivorus*
Photo 3: *Rattus rattus alexandrinus*
Photo 4: A closed jar containing cotton with diethyl ether for general anesthetic