

The occurrence and ultrastructure of *Trypanosoma* (*Herpetosoma*) *lewisi* (Kent, 1880) Laveran and Mesnil, 1901, the parasite of rats (*Rattus norvegicus*) in Poland

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ABSTRACT. This study reports the light and electron microscopic examination of *Trypanosoma* (*Herpetosoma*) *lewisi* (Kent, 1880) Laveran and Mesnil, 1901, isolated from rats (*Rattus norvegicus*) from Poland. Bloodstream trypomastigotes were identified morphometrically from 100 specimens collected from three naturally infected rats *Rattus norvegicus*. Body length ranged from 15.45–23.64 μm and width from 1.3–2.32 μm while the free flagellum was 8.1 μm long. Electron microscopic study of bloodstream trypomastigotes exhibited typical ultrastructural features similar to those of other stercorarian trypanosomes. The presently determined morphological data have been compared with those provided by other authors.

Key words: *Trypanosoma* (*Herpetosoma*) *lewisi*, *Rattus norvegicus*, Rodentia, Poland

Introduction

Due to the continuous urbanization and contraction of the area not transformed by human activity, many animal species adapted to the synantropic environment. Among them there are all components of zoonotic foci structure. The rodents, being the reservoir of many human and domestic animals pathogens. Their vectors – bloodsucking ectoparasites – become also the components of synantropic fauna [1]. In the urban areas, the most common rodents are rats – *Rattus norvegicus*, *Rattus rattus* and mouse (*Mus musculus*) [2]. Municipal recreational areas can also be inhabited by rodents typical for woodland areas, such as the bank vole, *Clethrionomys glareolus*, *Microtus* voles and *Apodemus* mice [3].

The ability of rats to acquire and spread of viral, bacterial and parasitic diseases of human and pets has been commonly known and well documented [2]. However, there are only few data available about parasites of no medical importance, as blood parasites, infecting synantropic rodents. They are frequently encountered, but the knowledge on their effect on the host's health and on their interactions with other parasites is weak.

Trypanosoma (*Herpetosoma*) *lewisi* (Kent, 1880) Laveran and Mesnil, 1901 is a parasite of *Rattus rattus* and *Rattus norvegicus*. The earliest description of this trypanosome, from *Rattus rattus*, dates from France, 1850. It was later reported in Austria, former Czechoslovakia, England, Russia and the USA [4,5]. In Poland, their occurrence in the blood of wild rats has been documented for the first time in Warszawa by Karbowiak and Wita [6]. It is a cosmopolitan species. The rats are commonly infected, however, the incidence of infections varies in different parts of the world, depending on the different ecological and zoogeographical conditions. However, the analysis of various reports shows that in the populations inhabiting this same locality, the incidence of *T. lewisi* in *R. norvegicus* is usually higher than that in *R. rattus*. The prevalence of *R. norvegius* infections range, depending on the season and locality, from 2.3 to 41%. The prevalence values *R. rattus*, however, are between 7% and 29% (Table 1). The infections of other *Rattus* species are known as well. Kartman [7] described the infection in *Rattus hawaiiensis*. Other mammals were reported as hosts [5], among them human [8], but the most recent sources reject the latter option [9].

The infection is observed in young individuals mainly, to 4 months of age [7,9]. This fact is caused, probably, by the possibility of infection of young animals by fleas as early as in the nest, and on the other hand, by the fact that rats acquire the resistance on the next trypanosome infection [10]. Moreover, the males are more commonly infected than females. It can be also justified by behavioral factors. Males penetrate wider areas than females and they are more active. Therefore males are more likely to pick up fleas and consequently trypanosome infection [10,11].

In temperature climate zone *T. lewisi* is transmitted by rat flea, *Nosopsyllus fasciatus*, while in tropical countries by oriental rat flea, *Xenopsylla cheopis*. Other possible vectors are: the dog flea, *Ctenocephalides canis*; human flea, *Pulex irritans*; mouse fleas, *Leptopsylla segnis*, *Ctenophthalmus assimilis*, and *Ceratophyllus hirundinus*. The fleas are important reservoir hosts for this parasite; the infected specimens carry the parasites throughout their entire life [9,12,13].

The infection of rat takes place from invertebrate vector through fecal contamination or orally by flea ingestion. The transmission of Herpetosoma trypanosomes by that way has been experimentally documented [14,15]. It is possible also the infection by the hurts, on example during the combats between rat individuals [16].

The course of infection in the rat is typical for parasitic protozoa and can be divided into three periods: the incubation stage; the reproductive (multiplication) stage; and the stage of „adult" infection. The incubation stage, since the inoculation of the metacyclic forms of parasite and the appearance of the trypanosomes in the blood, lasts 1–7 days. This time the metacyclic trypomastigotes transform into epimastigotes, which are the reproductively competent form of the parasite in the vertebrate hosts [5,9].

In the reproductive (multiplication) stage numerous divisions of parasites can be observed. Thus their number rapidly grows, to 300 000 individuals in 1 ml. This period lasts 10–25 days, and characterized by exxtensive variations of parasites in size and body proportion, due to the simultaneous occurrence of young, adults and divided individuals [9,17]. Contrary to commonly known Salivaria trypanosomes of *Nannomonas* and *Trypanozoon*, dividing into two young specimens, *Herpetosoma* Stercoraria produces 8–12 specimens. *T. lewisi* divides at the epimastigote stage.The division takes place in the blood, epimastigotes do not migrate to the internal organs. The young individuals are epimastigotes. They grow, and after reaching full size, they start the next dividing cycle, or transform in trypomastigote forms. Trypomastigotes are considered as adult stage, and they do not divide in mammal organism [12, 18].

The next period – adult stage – means the cessation of multiplication and the growth arrest. An impulse for the getting of infection from the pleomorphic reproductive stage into the not reproductive monomorphic stage is the acquirement of the resistance by the host. The immunological response consists in the combined action of ablastin and trypanocidal antibodies and it is directed against the surface antigens of epimastigote and the dividing forms. In the first period of this phase, the antibodies do not kill parasites, but block their multiplication and induce the transformation from the epimastigote form into the trypomastigote. Only in next stages epimastigotes, having no time left for transformation are killed. Adult trypomastigote forms remain intact, but their numbers in the blood also gradually decrease [18,19]. This period lasts from one to many weeks at the end of which time the trypanosomes completely disappear from the blood. Only in the case of latent infections few epimastigote forms remain hidden in capillary vessels and cells of the kidney's core and in the spleen [17,18,20].

Mammal sacquire permanent immunity, thus the remained trypomastigotes are not capable of restoring the parasite's population, and they can be only a source of the infection for bloodsucking invertebrates. It should be emphasized that, except immunological response, the increase of the trypanosomes number can be limited in the blood by the unknown internal regulatory mechanisms. They do not allow to the rapid increasing of the population of the parasite which would be able to threaten to the life of the host [21].

Materials and methods

Herpetosoma trypanosomes, used in this study, were isolated from the fresh blood of naturally infected rats, *Rattus norvegicus* in the Warszawa suburbs in Milanówek (3 individuals, 1♂ and 2♀, in this 2 infected 1♂ and 1♀) and in rats captured in the Deer Farm in Kosewo Górne, Mazurian District (5 individuals, 2 ♂ and 3♀, 1 ♂ infected). All infected rats were juveniles not exceeding 130 g of body weight. No clinical symptoms in infected animals were observed. Blood from rats was collected from the tip of their tails, using heparinised haematocrit

Table 1. The prevalence of *Trypanosoma lewisi* in *Rattus norvegicus* and *Rattus rattus* at various localities

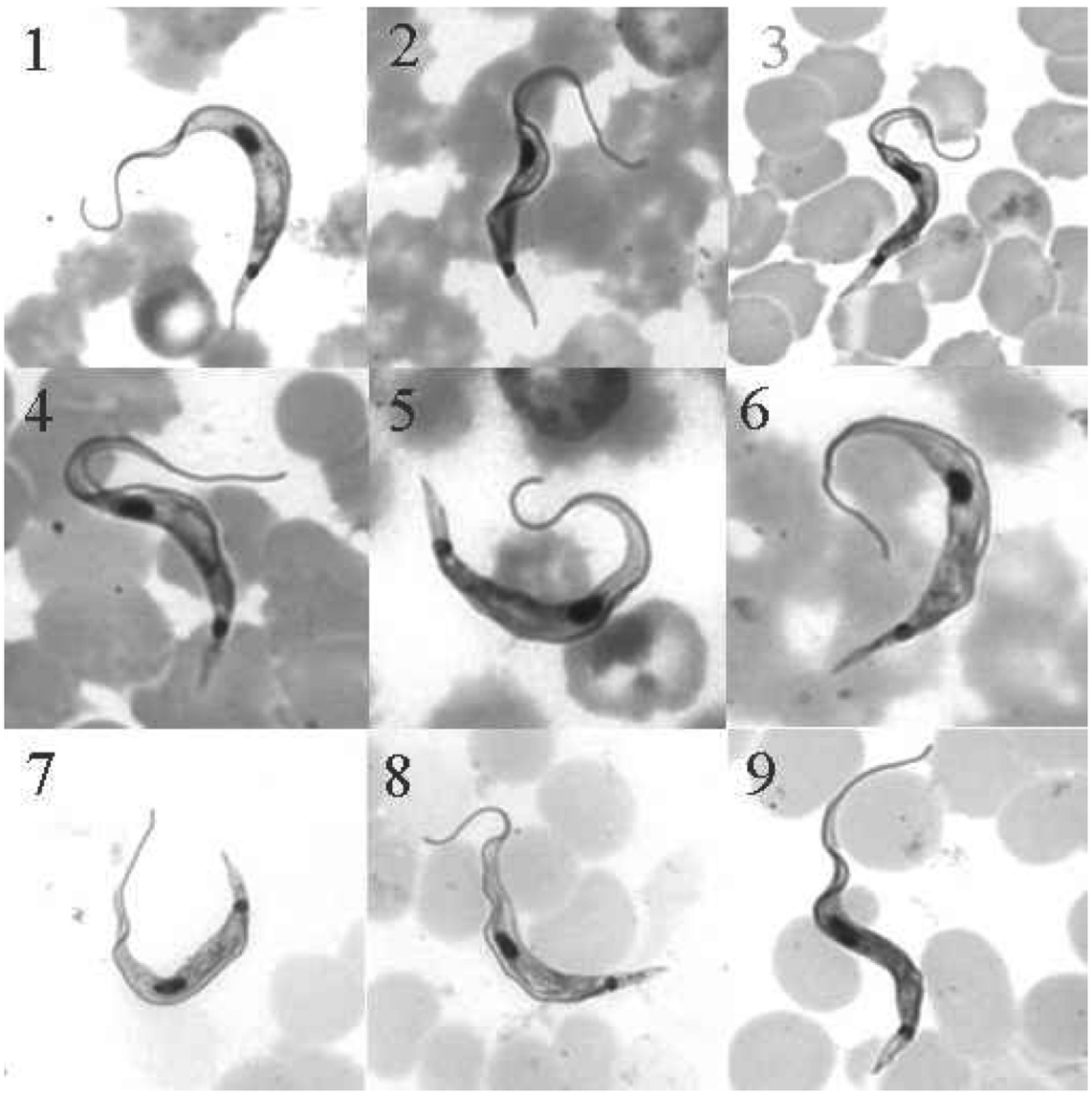
Rattus norvegicus		
Locality	Prevalence	Author
Khuzestan, Iran	10%	Kia et al. [31]
Minas Gerais, Brasil	21.7%	Linardi and Botelho [10]
Auckland, New Zealand	12–30%	Doré, after Kartman [7]
Wellington, New Zealand	4.6%	Laird, after Kartman [7]
Hamacua, Hawaii	11.4%	Kartman [7]
Baghdad, Iraq	2.27%	El-Shenawi, after Molan and Hussein [32]
Baghdad, Iraq	17.24%	Molan and Hussein [32]
Khuzestan, Iran	10%	Kia et al. [31]
Windam, India	41.2%	Saxena and Miyata [13]
Rattus rattus		
Locality	Prevalence	Author
Madagascar	11.5%	Laakkonen et al. [24]
Hamacua, Hawaii	22.2%	Kartman [7]
Maharashtra, India	28.9%	Mourya et al. [29]
Baghdad, Iraq	11.80–16.26%	El-Adhami, after Molan and Hussein [32]
Baghdad, Iraq	7.07%	Molan and Hussein [32]
Kuala Lumpur, Malaysia	21.7%	Zainal-Abidin and Noor [17]

tubes and subsequently examined for the presence of trypanosomes. The blood smears were fixed with methyl alcohol and stained with Giemsa. Blood smears were scanned at a magnification of 1200× by light microscopy coupled with a computer and video camera, using an Analysis Pro 2.11 program. Morphometric observations followed the nomenclature and methodology of Hoare [5]. One hundred adult, trypomastigotes were measured. Measurements and calculated indices were compared with those of *T. lewisi* from *R. norvegicus* and from *R. rattus* given by other authors (Table 2 and 3).

The trypanosomes intended for transmission electron microscopy were fixed in 2.5% (w/v) glutaraldehyde solution in 0.05 M sodium cacodylate buffer, pH 7.4, containing 0.12 M sucrose and 5 mM $CaCl_2$. After washing in cacodylate buffer and postfixation in 2.0% (w/v) osmium tetroxide in cacodylate buffer for 1 h at 4°C, the specimens were rinsed and dehydrated through an ascending series of ethanol transferred to absolute acetone and embedded in Epon resin. Ultrathin sections were stained using uranyl acetate and lead citrate [22]. Micrographs were taken with a JEM 100B electron microscope (80 kV).

Results

The trypanosomes found were mainly in trypomastigote stage (Figs. 1–9). There were also few epimastigotes. The morphological features and size of trypomastigotes in all localities were similar. Body length was 15.45–23.64 μm, width 1.3–2.32 μm. The undulating membrane was moderate developed. The free flagellum was 4.93–11.51 μm (mean 8.09) long. The nucleus was situated at the anterior part of the cell body (NI=1.59). Kinetoplast was oval and located nearer to the posterior end of the body than to the nucleus. The body size and morphology was also similar to data given by other authors (Table 2).



Figs. 1–9. Light micrographs of Giemsa-stained bloodstream trypomastigotes of *Trypanosoma* (*Herpetosoma*) *lewisi* from *R. norvegicus* in Poland. *T. lewisi* has a long thin posterior end, with a subterminal ovale kinetoplast, the nucleus is in the anterior part of the body flagellum is free. Scale bar=10 μm.

Bloodstream forms of *Trypanosoma lewisi* from *Rattus norvegicus* ultrastructurally are similar to bloodstream forms of other trypanosomatid parasites [5, 20, 23–26]. They are bounded by a plasma membrane 8–10 nm thick (Fig. 13). A surface coat was present on the plasma membrane. Its thickness was variable at different parts of the body (4–19 nm). This is consistent with the results of previous ultrastructural studies of stercorarian trypanosomes such as *T. cruzi* [27, 28]. The cytoskeleton is characterized by a subpellicular corset of microtubules, 25 nm in diameter. They are located 7–11 nm below the plasma membrane, the distance between the microtubules is 13–25 nm (Figs. 12, 13). In the area where the flagellum extends through the plasma membrane one or more microtubules may be lacking. It was observed that the number of microtubules is related to the diameter of the cell. At the posterior and anterior regions of the trypomastigotes the number of microtubules is smaller.

Rod-like kinetoplast associated with a mitochondrion was located close to the posterior end of the trypomastigote body (Figs. 1–9) and far from the posterior end in epimastigote forms (Figs. 10, 14). The single mitochondrion with well developed plate-like cristae formed peripherally located canal running the length of the cell body (Fig. 17a). In nucleus a prominent, centrally located nucleolus and peripheral chromatin are seen (Figs. 15, 17a). It was observed that *T. lewisi* divided as epimastigotes

Table 2. Dimensions (μm) of *Trypanosoma lewisi* trypomastigotes from *Rattus norvegicus* (by various authors)

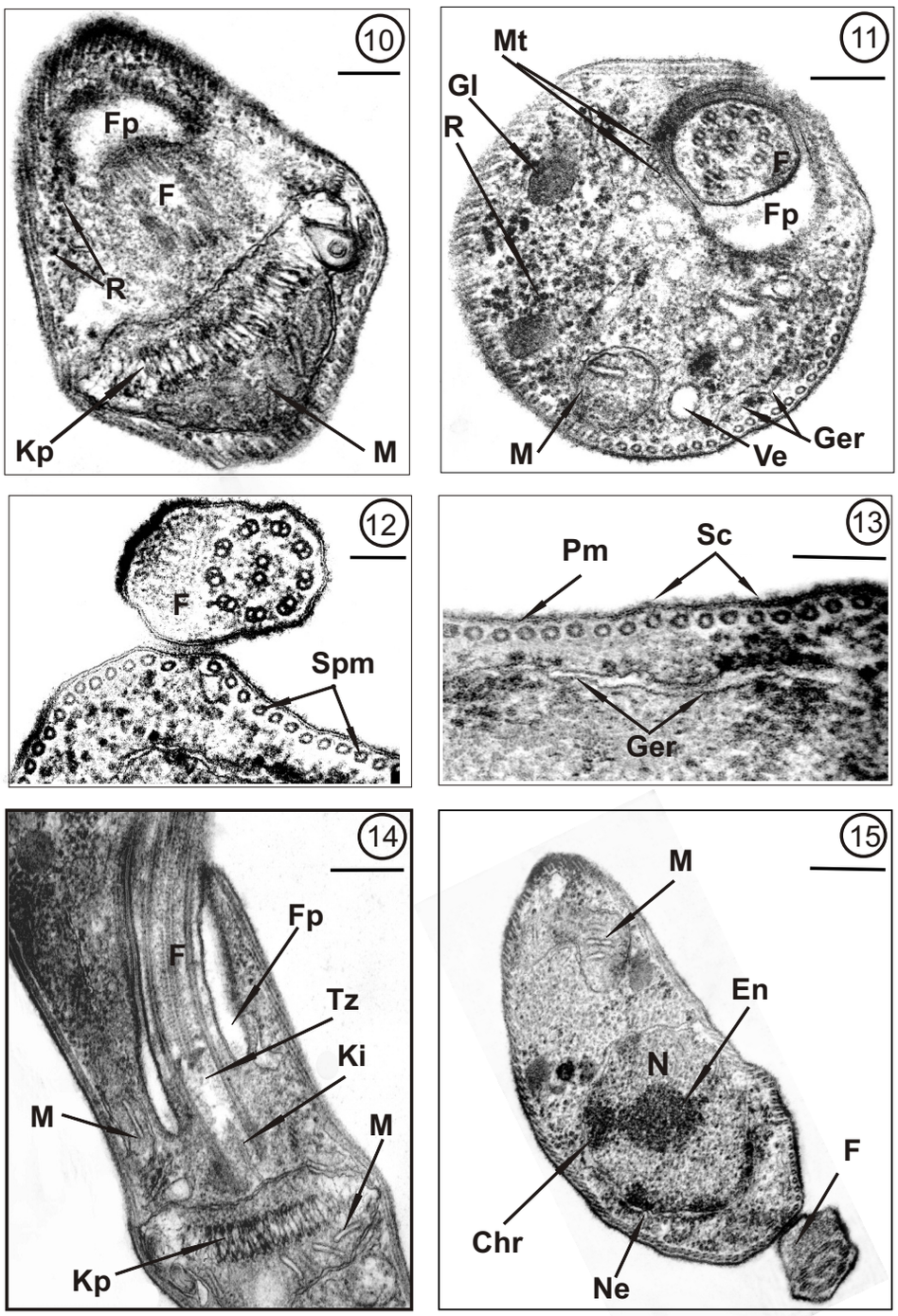
		T. lewisi by Karbowiak and Wita [6]	*T. lewisi* by Taliaferro [4] Baltimore, USA	*T. lewisi* by Kartman [7] Island of Hawaii	*T. lewisi* by Mansfield [18]
PK	mean±SD	3.01±0.70	4.27±0.04		
	range	1.11–4.33	–		
KN	mean ±SD	8.93±0.61	10.85±0.02		
	range	7.27–10.24	–		
PN	mean±SD	11.94±0.78	15.12*	15.0	
	range	9.84–13.38			
NA	mean±SD	7.82±1.54	9.51±0.05	8.8	
	range	4.42–10.65	–	–	
BL	mean±SD	19.76±1.97	24.63	23.8	23.1
	range	15.45–23.64	–		
FF	mean±SD	8.09±1.51	6.62±0.07	7.4	7.5
	range	4.93–11.51	–	–	7.2–7.8
L	mean±SD	27.84±1.30	31.25±0.06	31.2	30.6
	range	24.46–30.39	–	–	21.0–36.5
N	mean±SD	2.11±0.28		2.1	
	range	1.30–2.75		–	
W	mean±SD	1.77±0.24	1.59±0.02	1.9	–
	range	1.30–2.32	–	–	1.5–2.2
KI	mean±SD	1.34±0.25	1.39		1.5
	range	1.13–1.50	–		–
NI	mean±SD	1.59±0.33	1.59	1.7	–
	range	1.11–2.58	–		1.2–2.0
FF:BL	mean±SD	2.56±0.67	3.72		3.08
	range	1.43–4.26	–		

*Shadow fields – calculated here on the basis of original data
Explanations: PK – distance between posterior end of body and kinetoplast; KN – distance between kinetoplast and nucleus centre; PN – distance between posterior end of body and nucleus centre; NA – distance between nucleus centre and anterior end of body; BL – body length; FF – length of free flagellum; L – total length; W – width of body; N – length of nucleus; NI – nucleus index (NI=PN/NA); KI – kinetoplastic index (KI=PN/KN); FF:BL – flagellar index (=BL/FF).

(Figs. 17a, b, c, d) . The Golgi apparatus is situated at the anterior part of the cell body. It was composed of 7–8 flattened cisternae (Fig. 16). Small vesicles were observed in the region of the Golgi apparatus. The rough endoplasmic reticulum was well developed forming a characteristic cisterns (Figs. 13, 16). Free ribosomes were numerous in the cytoplasm (Fig. 11). The vacuoles and small vesicles are seen throughout the cytoplasm (Fig. 11). They differ in shape, dimensions and electron density. Coated glycosomes with an electron dense matrix were scattered throughout the cytoplasm (Figs. 11, 16). The cytoplasm also contained large digestive vacuoles. Structure of the kinetosome, flagellar pocket and flagellum do not differ from the type described in bloodstream stercorarian trypomastigotes.

Discussion and general conclusions

Analysis of the light microscopic and ultrastructural data confirmed that the parasites found in the blood of the *Rattus norvegicus* in Poland were *Try-*



Figs. 10–17. Transmission electron micrographs of *Trypanosoma* (*Herpetosoma*) *lewisi* – bloodstream trypanosomes.
Figs. 10–11. Details of the oblique (10) and transverse (11) section of epimastigote form showing the flagellum, kinetoplast, mitochondrion. Glycosomes and membrane bound vesicles in the flagellar pocket region also are visible. The cytoplasm is filled with numerous free ribosomes. Scale bar=0.2 μm.
Figs. 12–13. Section of the flagellum and of the part of surface of trypomastigote form showing subpellicular microtubules reinforcing the plasma membrane which bears a surface coat. Scale bar=0.1 μm.
Fig. 14. Longitudinal section of anterior region of the epimastigote showing details of the flagellum, transitional zone and kinetosome. The kinetoplast in the expanded part of the mitochondrion is observed in close association to kinetosome. Scale bar=0.3 μm.
Fig. 15. The nucleus with the central endosome, peripheral chromatin and double nuclear envelope is demonstrated. Scale bar=0.3 μm.

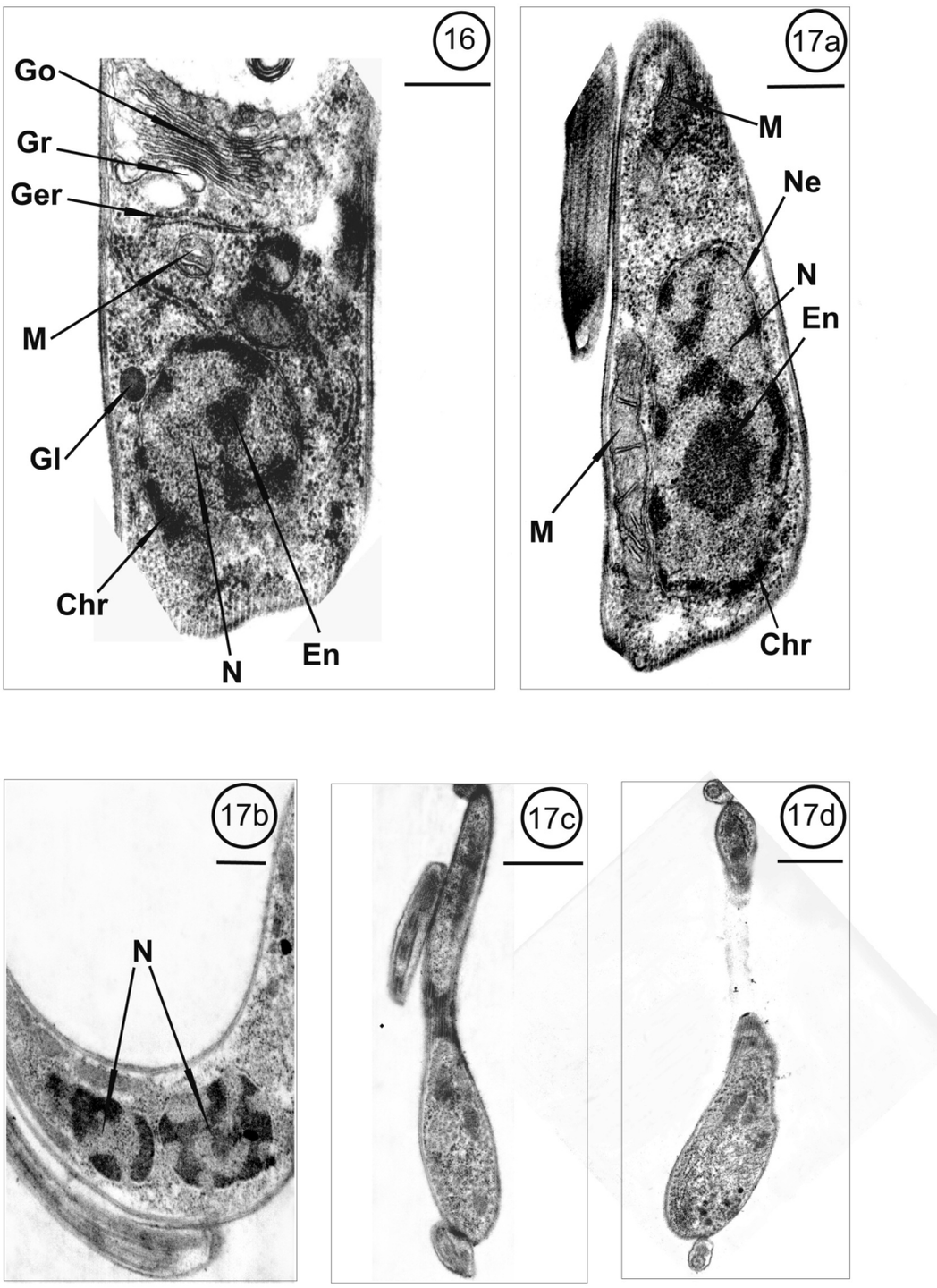


Fig. 16. Section through the trypanosome showing the glycosome (Gl), granular endoplasmic reticulum (Ger) and Golgi apparatus with elongate cisternae and Golgi vesicles are demonstrated. Scale bar=0.5μm.
Figs. 17a, b, c, d. Section of dividing forms of epimastigote. Abbreviations as for other figures. Scale bar=0.5 μm for Figs. 17a, b. Scale bar=1.0 μm for Figs. 17c, d.
Abbreviations for all figures **Chr** – chromatin, **En** – endosome, **F** – flagellum, **Fp** – flagellar pocket, **Ger** – granular endoplasmic reticulum, **Gl** – glycosome, **Go** – Golgi apparatus, **Gv** – Golgi vesicle, **Ki** – kinetosome, **Kp** – kinetoplast, **M** – mitochondrion, **Mt** – microtubules, **N** – nucleus, **Ne** – nuclear envelope, **Np** – nuclear porc, **Pm** – plasma membrane, **R** – free ribosomes, **Sc** – surface coat, **Tz** – transitional zone, **Ve** – membrane bound vesicle, **Spm** – subpellicular microtubules.

Table 3. Dimensions (μm) of *Trypanosoma lewisi* trypomastigotes from *Rattus rattus* (by various authors)

	Indices	*T. lewisi* by Kartman [7] Island Hawaii	*T. lewisi* by Mourya et al. [29] India	*T. lewisi* by Laakkonen et al. [24] Madagascar
PK	mean			4.95
	range			4.70–5.50
KN	mean			9.10
	range			8.65–9.75
PN	mean	14.40*		14.00
	range	–		13.50–14.55
NA	mean	8.3		5.17
	range	–		4.35–7.05
BL	mean	22.70		19.20
	range	–		17.9–21.20
FF	mean	7.3		6.06
	range	–		6.00–6.25
L	mean	30.00	35.6	25.26
	range	–	–	23.85–27.20
N	mean	2.1		2.97
	range	–	2.87–3.00	
W	mean	1.1		1.76
	range	–		1.68–1.90
KI	mean			1.55
	range			1.49–1.64
NI	mean	1.73		2.82
	range	–		2.01–3.16
FF:BL	mean	3.11		3.17
	range	–		2.98–3.53

*Shadow fields – calculated here on the base of data publicated on the basis of original data. Explanations: see Table 2.

panosoma lewisi. It was not possible to compare of all morphometric values of bloodstream trypomastigotes from *R. norvegicus* from Poland with those data obtained by other authors for *T. lewisi* from *R. norvegicus* and *R. rattus* (see accompanying Tables 2 and 3).

The mean values of PK, KN, PN, NA, BL, L and KI parameter in trypanosomes from *Rattus norvegicus* from Poland were smaller that the mean values given by Taliaferro [4], Kartman [7] and Mansfield [18] (Table 2). Only the average free flagellar length (FF) was slightly higher and FF:BL ratio lower in trypanosomes from Poland. The mean length of the nucleus was the same for trypanosomes from Poland and from Hawaii [7]. The value of the nuclear index (NI=1.59) was identical as for trypanosomes from USA and smaller (NI=1.70) than calculated for trypanosomes from Hawaii. Mansfield [18] reported on collective group of trypanosomes gave only the range of NI index without mean value. These values of the nuclear index showed that nuclei of detected trypanosomes were located in the anterior part of the body. The kinetoplast in trypanosomes from *R. norvegicus* from Poland was at a smaller distance (mean 3.01 μm) from the posterior end (PK) than in trypanosomes (mean 4.27 μm) from *R. norvegicus* from USA [4]. Also kinetoplastic index (KI), used to define the position of kinetoplast, derived

from our trypanosomes was lower than calculated for trypanosomes from USA [4] (mean 1.39) and for colective group of *T. lewisi* [18] (mean 1.5) and showed that all compared specimens had a kinetoplast located near the posterior end of the body.

When the morphometric values of *T. lewisi* from *R. norvegicus* [4,6,7,18] (Table 3) were compared with the same parameters of *T. lewisi* from *R. rattus* [7,24,29] (Table 3) we revealed many similarities and some differences in morphometric measurements of trypanosomes from these different hosts. The mean values of PK, KN, PN, N, KI and NI parameters in trypanosomes from *R. norvegicus* from Poland were smaller that these mean values given by Laakkonen et al. [24] for trypanosomes from *R. rattus* (Table 3). The value of the nuclear index (NI=1.59) showed that nuclei in our specimens were located more posteriorly than nuclei in *T. lewisi* from *R. rattus* (NI=2.82). Kinetoplastic index (1.34 and 1.55, respectively) showed that all specimens had a kinetoplast near the posterior end of the body. The average free flagellar length (FF) was higher and FF:BL ratio lower in trypanosomes from Poland. Our results showed higher mean values of NA (7.86 μm) and L (25.26 μm) than have been found in *R. rattus* by Laakkonen et al. [24]. The mean length (BL) and width of the body (W) were similar for trypanosomes from both host species.

Kartman [7] reported on trypanosomes from *R. rattus* in Island Hawaii. The mean values of PN, NA and BL given by Kartman [7] were higher than the mean values for trypanosomes from *R. norvegicus* from Poland and smaller than given by Taliaferro [4], Kartman [7] and Mansfield [18] also for trypanosomes from *R. norvegicus.* The mean of total length (L) of *T. lewisi* from *R. rattus* [7] was slightly higher than the mean value for investigated by us trypanosomes and almost the same as the data given by Taliaferro [4], Mansfield [18] and Kartman [7] for *R. norvegicus.* In our opinion there are no significant differences in L and BL values in trypanosomes *T. lewisi* from *R. norvegicus* and *R. rattus* because differences in the range of length measurements were not seen. Only the average free flagellar length (FF) was slightly lower and FF:BL ratio higher in trypanosomes from Poland. The mean length of the nucleus was the same for trypanosomes from both host species. The value of the nuclear index (NI=1.73) showed that nuclei in trypanosomes from Island Hawaii were located more anteriorly than nuclei in our specimens (NI=1.59).

Mourya et al. [29] reported on trypanosomes from *R. rattus* in India gave only the mean of value for body length (36.6 μm). This value is higher than values of body length, given by other authors [4, 6, 7,18,24] for *T. lewisi* from *R.norvegicus* and *R. rattus.*

The results of our investigations show that trypanosomes parasitizing the *R. norvegicus* from Poland appear to be smaller than *T. lewisi* from *R. norvegicus* [4,7,18] and from *R. rattus* [7,24,29]. In our opinion there are no significant differences in morphometric values given in the descriptions of *T. lewisi* by several authors. Trypanosomes from the rats show considerable plasticity in morphometric characters although the small differences in measurements reported by different authors may be related to the techniques used, and to the stage of infection when the organisms were examined, i.e., chronic or recent infection.

Our investigation of the fine structure of *T. lewisi* from *R. norvegicus* from Poland exhibited typical trypanosome ultrastructure [5,20,23,24,30] and indicated a strong similarity between this trypanosome and other stercorarian species.

In our survey of endoparasites of rats, we found that trypanosome *T. lewisi* is a cosmopolitan species and a quite common parasite of *Rattus norvegicus* in Poland. No signs of pathogenicity were observed in rats infected with trypanosomes. The observations of trypomastigote forms of *T. lewisi* living in the host's blood confirmed low pleomorphism of this parasite [6,9]. The pleomorphism is probably more linked to variability of developmental forms than differences between different strains. In many cases the morphological differentiation is in correlation with the age of trypanosomes. Taliaferro [4] showed, that variations in the size of single strains adults specimens are statistically insignificant.

References

[1] Černý V., Rosický B. 1979. Mammals as source of ectoparasites in towns. *Folia Parasitologica* 26: 93–95.

[2] Wincewicz E., Klimentowski S., Jopek Z., Śmielewska-Łoś E., Szarycz M. 1999. Rola szczurów z epizootiologicznego i epidemiologicznego punktu widzenia. *Medycyna Weterynaryjna* 55: 234–238.

[3] Karolewski M.A. 1981. Specyfika i status ekologiczny miasta. *Wiadomości Ekologiczne* 27: 3–35.

[4] Taliaferro W.H. 1921. Variation and inheritance in size in *Trypanosoma lewisi*. *Proceedings of the National Academy of Sciences of the United States of America* 7: 138–143.

[5] Hoare C.A. 1972. The trypanosomes of mammals. Blackwell Scientific Publications, Oxford, Edinburgh.
[6] Karbowiak G., Wita I. 2001. Przypadki zarażenia szczurów wędrownych *Rattus norvegicus* (Berkenhout, 1769) świdrowcem *Trypanosoma lewisi* (Kent, 1880) Laveran i Mesnil, 1901 na terenie aglomeracji warszawskiej. *Wiadomości Parazytologiczne* 47: 377–382.
[7] Kartman L. 1954. Observations on *Trypanosoma lewisi* and *Grahamella* sp. in the blood of rats from the Hamakua district, Island of Hawaii. *Journal of Parasitology* 40: 571–579.
[8] Shrivastava K.K., Shrivastava G.P. 1974. Two cases of *Trypanosoma* (*Herpetosoma*) species infection of man in India. *Transactions of Royal Society of Tropical Medicine and Hygiene* 68: 143–144.
[9] Desquesnes M., Ravel S., Cuny G. 2002. PCR identification of *Trypanosoma lewisi*, a common parasite of laboratory rats. *Kinetoplastid Biology and Disease* 1: 2.
[10] Linardi P.M., Botelho J.R. 2002. Prevalence of *Trypanosoma lewisi* in *Rattus norvegicus* from Belo Horizonte, State of Minas Gerais, Brazil. *Memorias do Instituto Oswaldo Cruz* 97: 411–414.
[11] Linardi P.M., Botelho J. R., Cunha H.C. 1985. Ectoparasitos de roedores da região urbana de Belo Horizonte, MG. II. Oscilações dos índices de infestação em *Rattus norvegicus norvegicus. Memorias do Instituto Oswaldo Cruz* 80: 227–232.
[12] Molyneux D.H. 1970. Developmental patterns in trypanosomes of the subgenus *Herpetosoma*. *Annales de la Societe Belge de Médecine Tropicale* 50: 229–238.
[13] Saxena V.K, Miyata A. 1993. An unusual morphological type of *Trypanosoma* (*Herpetosoma*) *lewisi* (Kent, 1880) detected in the blood of *Rattus norvegicus* in India. *Journal of Communicable Diseases* 25: 15–17.
[14] Clarkson M.J., McCabe W.J. 1973. Oral transmission of trypanosomes. *Transactions of Royal Society of Tropical Medicine and Hygiene* 67: 12.
[15] Maraghi S., Wallbanks K.R., Molyneux D.H. 1995. Oral transmission of trypanosomes of the subgenus *Herpetosoma* from small mammals. *Parasitology Research* 81: 693–695.
[16] Molyneux D.H. 1976. Biology of Trypanosomes of the subgenus *Herpetosoma*. In: *Biology of the Kinetoplastida*. (Eds. W.H.R. Lumsden, D.A Evans). Academic Press, London, New York, San Francisco: 285–325.
[17] Zainal-Abidin B.A.H., Noor A.A. 1999. Jangkitan *Trypanosoma lewisi* Pada Tikus Liar. *Sains Malaysiana* 28: 1–8.
[18] Mansfield J.M. 1977. Nonpathogenic trypanosomes of mammals. In: *Parasitic Protozoa 1*. (Ed. J.P. Kreier). Academic Press, New York: 297–327.
[19] Albright J.W., Albright J.F. 1991. Rodent trypanosomes: their conflict with the immune system of the host. *Parasitology Today* 7: 137–140.
[20] Greenblatt C.L. 1973. *Trypanosoma lewisi*: electron microscopy of the infected spleen. *Experimental Parasitology* 34: 197–210.
[21] Vassella E., Schulte zu Sodingen C., Wild N., Reuner B., Klöckner T., Krämer R., Wankell M., Boshart M. 1998. Control of proliferation and differentiation of *Trypanosoma brucei* bloodstream forms. *Parasitology International* 47 (Suppl.): 221.
[22] Reynolds E.S. 1963. The use of lead citrate at high pH as an electron-opaque stain in electron microscopy. *Journal of Cell Biology* 17: 208–212.
[23] Burton P.R., Dusanic D.G. 1968. Fine structure and replication of the kinetoplast of *Trypanosoma lewisi*. *Journal of Cell Biology* 39: 318–331.
[24] Laakkonen J., Goodman S.M., Duchemin, J-B., Duplantier J-M. 2003. Trypomastigotes and potential flea vectors of the endemic rodents and the introduced *Rattus rattus* in the rainforests of Madagascar. *Biodiversity and Conservation* 12: 1775–1783.
[25] Soares M.J., Souza W. 1988. Cytoplasmic organelles of trypanosomatids: a cytochemical and stereological study. *Journal of Submicroscopic Cytology and Pathology* 20: 349–361.
[26] Vickerman K., Brugerolle G., Mignot J-P. 1991. Mastigophora. In: *Microscopic Anatomy of Invertebrates. Volume 1: Protozoa*. (Eds. F.W. Harrison, J.O. Corlis). New York, Wiley-Liss: 13–159.
[27] Brener Z. 1973. Biology of *Trypanosoma cruzi. Annual Review of Microbiology* 27: 347–382.
[28] Almeida I.C., Ferguson M.A.J, Schenkman S., Travassos L.R. 1994. Lytic anti-α-galactosyl antibodies from patients with chronic Chagas' disease recognize novel O-linked oligosaccharides on mucin-like glycosyl-phosphatidylinositol-anchored glycoproteins of *Trypanosoma cruzi*. *Biochemical Journal* 304: 793–802.
[29] Mourya D.T., Geevarghes G., Gokhale M.D. 1996. Note on the occurrence of *Trypanosoma* (*Herpetosoma*) sp. in *Rattus rattus* in Beed district, Maharashtra. *Journal of Communicable Diseases* 28: 299–300.
[30] Simaren J.O. 1973. Ultrastructure of *Trypanosoma lewisi*, localization and alterations in rat liver. *Annales de Parasitologie Humaine et Comparée* 48: 735–754.
[31] Kia E.B., Homayouni M.M., Farahnak A., Mohebali M., Shojai S. 2001. Study of endoparasites of rodents and their zoonotic importance in Ahvaz, South West Iran. *Iranian Journal of Public Health* 30: 49–52.
[32] Molan A.L., Hussein M.M. 1988. A general survey of blood and tissue parasites of some rodents in Arbil province, Iraq. *APMIS* (Suppl. 3): 47–49.

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