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# PATHOGENICITY AND ULTRASTRUCTURAL PATHOLOGY OF EIMERIA DEBLIECKI (DOUWES, 1921) IN EXPERIMENTALLY INFECTED PIGS

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**Abstract.** It was confirmed, after experimental infection of 24 weaned pigs with different doses (200,000 and 4 mil.) of *Eimeria debliecki* oocysts that the developmental cycle of *E. debliecki* occurs in the anterior jejunum and after a high dose of oocysts also in the duodenum and anterior parts of the middle jejunum. Pathological changes characterized by a light atrophy of the villi, scarcely dispersed minute erosions of the epithelium in upper parts of the mucosa and an inflammatory response in the propria of the anterior jejunum were found in the area of the largest occurrence of developmental stages of *E. debliecki* (from 50 cm to 100 cm from the pylorus). An inflammatory infiltrate in the propria of the anterior jejunum from 3 DPI to 5 DPI contained a conspicuously large number of plasma cells with Russell bodies. Cellular changes were detected only in enterocytes with developmental stages of *E. debliecki*. Changes of the microvillous zone were observed in infected enterocytes; dilated mitochondria, free ribosomes and an increased number of residual bodies were found in the cytoplasm of enterocytes. The degree of the cellular changes of enterocytes was dependent on the maturity of the developmental stage of *E. debliecki*. Based on pathological changes observed by the use of light and electron microscopy, the coccidium *E. debliecki* is considered to be pathogenic for weaned pigs in spite it does not provoke a clinical infection.

Results of coprological examinations of pigs show that the coccidium *Eimeria debliecki* has cosmopolitan distribution and is the most frequent coccidium of pigs (Kutzer 1960, Boch et al. 1961, Vetterling 1966a, Romero and Liozano 1971, Lindsay et al. 1984, Arnastauskiene 1985, Varghese 1986, Horváth and Nemes 1988).

Opinions on pathogenicity of the coccidium *Eimeria debliecki* vary. Boch and Wiesenhutter (1963) have infected 2-7-week-old piglets with 20,000—50,000 oocysts of *E. debliecki* and in three piglets infected with a dose of 50,000 oocysts a diarrheal disease have been found. At necropsy, these authors have found catarrhal inflammatory changes in the middle and posterior jejunum. On the basis of these results they consider the coccidium *E. debliecki* to be pathogenic. In contrast, Vetterling (1966b) in experimental infection of 2-week-old piglets with a dose of $2 \times 10^6$ oocysts of *E. debliecki* has not found clinical signs of the disease nor macroscopic pathological changes. Lindsay et al. (1987) consider *E. debliecki* to be a nonpathogenic species of coccidium which does not cause diarrheal disease in experimental infection of nursing and weaned pigs.

This paper gives results of histopathological and electron-microscopic examination of the intestine of pigs after experimental infection with the coccidium *E. debliecki*.

## MATERIALS AND METHODS

**Experimental animals.** A total of 24 weaned pigs 2-3-month-old were experimentally infected. All weaned pigs were hybrids of Landrace and Large White pig. The pigs were kept in sites in groups of 4, fed a commercial mixture and water ad libitum. Before the infection pigs were repeatedly coprologically examined.

**Oocysts of *Eimeria debliecki*.** Oocysts of *E. debliecki* were obtained from spontaneously infected

weaned pigs in the feces of which two species of coccidia were detected: *E. debliecki* and *E. neodebliecki*. After experimental infection of two weaned pigs with the mixture of these two coccidia, massive shedding of oocysts of *E. debliecki* was found from 6 to 8 day post infection (DPI). Oocysts of *E. neodebliecki* were not observed in the feces of these weaned pigs before 12 DPI.

Initially, only 16 weaned pigs (from ED 1 to ED 16) were infected with the obtained strain *E. debliecki*. From feces of two weaned pigs of this group (ED 6 and ED 7) further oocysts of *E. debliecki* were obtained and those were infected to the second group of weaned pigs (from ED 17 to ED 24).

**Method of experimental infection.** Suspension of oocysts of *E. debliecki* was stored in 2.5% potassium dichromate solution at 4 °C. Sporulated oocysts of *E. debliecki* used in experiments were as a maximum 2 months old. Before the infection the oocysts were washed repeatedly in PBS and number of oocysts in an infective dose was determined using Burker's chamber. Pigs were infected using an esophagus tube.

Survey of infections of pigs and number of *E. debliecki* oocysts in an infective dose are given in Table 1. Pigs used for multiplication of *E. debliecki* oocysts are not included in this table.

**Examination of experimental animals.** Daily clinical status of health was observed and the feces were taken for coprological examination in infected weaned pigs. Pigs were killed at various intervals post infection. Days of killing are summarized in Table 1.

**Table 1.** Survey of infections of gilts with the coccidium *Eimeria debliecki*

| Pig no. | Number of oocysts in infective dose | Day of killing after infection (DPI) |
|---|---|---|
| ED 1 | $4 . 10^6$ | 1 |
| ED 2 | $4 . 10^6$ | 2 |
| ED 3 | $4 . 10^6$ | 3 |
| ED 4 | $4 . 10^6$ | 4 |
| ED 5 | $4 . 10^6$ | 5 |
| ED 6 | $4 . 10^6$ | 6 |
| ED 7 | $4 . 10^6$ | 7 |
| ED 8 | $200 . 10^3$ | 3 |
| ED 9 | $200 . 10^3$ | 4 |
| ED 10 | $200 . 10^3$ | 5 |
| ED 11 | $200 . 10^3$ | 6 |
| ED 12 | $200 . 10^3$ | 7 |
| ED 13 | $200 . 10^3$ | 8 |
| ED 14 | $200 . 10^3$ | 9 |
| ED 15 | $200 . 10^3$ | 10 |
| ED 16 | $200 . 10^3$ | 11 |
| ED 17 | $200 . 10^3$ | 3 |
| ED 18 | $200 . 10^3$ | 3 |
| ED 19 | $200 . 10^3$ | 4 |
| ED 20 | $200 . 10^3$ | 4 |
| ED 21 | $200 . 10^3$ | 5 |
| ED 22 | $200 . 10^3$ | 5 |
| ED 23 | $200 . 10^3$ | 6 |
| ED 24 | $200 . 10^3$ | 6 |

After killing, samples for histological examination, scanning electron microscopy (SEM) and transmissive electron microscopy (TEM) were taken.

(A) **Histological examination.** Samples for histological examination were taken immediately after killing of pig. First of all, sample of th duodenum was taken, the following samples were taken from the anterior jejunum 50, 100 and 150 cm from the pylorus. Then, samples were taken every 100 cm in the caudal direction, so that the ileum close to the ostium ileocecale was the last taken portion of the small intestine. From the large intestine samples of the apex of the cecum, colon from the area of ansa centralis and rectum were taken. Then, samples of various organs from abdominal and thoracic cavity of pigs — the liver, kidney, spleen, mesenteric lymph-node, pancreas, lung and heart — were taken for histological examination. The samples were fixed in 10% neutral formalin. The material for histological examination was processed in usual manner with paraffin method. The histological sections were stained with hematoxylin eosin and azure eosin.

(B) **Scanning electron microscopy of the inner surface of the intestine.** Samples for SEM were taken immediately after killing from the same portions of the intestine as the samples for histological examination. The material for SEM was fixed in 4% buffered paraformaldehyde. Small strips of fixed intestinal mucosa were several times washed in distilled water, then, dehydrated in ascending alcohol series and in acetone and dried with method of critical point under carbon dioxide. Samples sputter coated with gold were observed in a TESLA BS 30 scanning electron microscope.

(C) **Transmissive electron microscopy (TEM) of the intestinal mucosa.** After TEM samples of the duodenum and anterior jejunum of pigs, from ED 17 to ED 24, were taken (from 3 DPI to 6 DPI). Samples for TEM were fixed in a paraformaldehydeglutaraldehyde fixative (Karnovsky 1965). The samples fixed in this way were subsequently fixed in 1 % $OsO_4$ (osmium oxide). Dehydrated samples were mounted in resin. Semithin sections were prepared in a LKB 3 ultramicrotome and stained with toluidine blue and according to Warmke and Sheu-Ling (1976). From selected samples ultrathin sections were made and observed in a Philips EM 420 transmissive electron microscope.



**Parasitological examination.** Fecal samples of infected weaned pigs were examined by flotation in Sheater's flotation solution (500 g of sugar, 6.5 g of phenol, 320 ml of water).

At necropsy, scrapings of the intestinal mucosa were taken from individual portions of the intestine, Giemsa-stained and occurrence of developmental stages of coccidia was estimated.

At necropsy of pigs, routine bacteriological examination of organs of abdominal and thoracic cavity was carried out which excluded specific bacterial pathogens of pigs.

**Control animals.** In the same way as experimentally infected pigs, 4 control pigs were examined. As control animals hybrids of Landrace and Large White pig 78-day-old were used.

## RESULTS

In the course of experimental infection with the coccidium *E. debliecki* in comparison with control animals no changes of clinical status of infected pigs were observed. Only from 3 DPI to 6 DPI increased content of mucus was observed in the formed feces of pigs infected with high doses of *E. debliecki* oocysts.

### Macroscopic changes

From 4 DPI to 5 DPI the wall of the anterior jejunum was slightly thickened, mucosa was reddish and edematous. Regional lymph nodes were slightly increased and edematous.

### Histopathological changes and changes of the inner surface of the intestine in SEM

At the first two days post infection no pathological changes were found in the intestine. The following day post infection (3 DPI) in pigs infected with high doses of oocysts ($4 \times 10^6$) meronts were observed in epithelium of the anterior jejunum and proximal part of the middle jejunum (Pl. I, Fig. 1). In the lamina propria of the same portions of the intestine a discrete inflammatory infiltrate with numerous eosinophils and plasma cells, which contained highly accumulated eosinophilic droplets was observed. Changes of the inner surface of the anterior jejunum did not occur at that time post infection.

At 4 DPI in the anterior jejunum and proximal part of the middle jejunum, focal invasion of epithelium with meronts was observed with maximum of changes from 50 to 100 cm from the pylorus. These meronts were found near tips of slightly atrophied villi, epithelium with developmental stages of the coccidium underwent metaplasia (Pl. I, Figs. 2, 3). The lamina propria of the anterior jejunum was edematous, hyperemic, permeated with inflammatory infiltrate which contained lymphocytes, neutrophils, eosinophils and macrophages. Apparently numerous were plasma cells. Some plasma cells possessed capacious, slightly eosinophilic cytoplasm and pyknotic nuclei near the periphery. In the cytoplasm of other plasma cells densely accumulated Russell bodies consisting of homogeneous eosinophilic droplets (Pl. IV, Fig. 1). In some plasma cells bodies were so accumulated that plasma cells became mulberry-shaped. Russell bodies were more frequent in the lamina propria of the middle and basal portion of mucosa. During examination of the inner surface of the intestine at 4 DPI slight villous atrophy was observed in the anterior jejunum; at the tips of villi and mucosal folds, scarce distributed minute erosions were found. In the lamina propria inflammatory infiltrate of the same character as at 4 DPI was observed.

During examination of the inner surface of the intestine slight villous atrophy with minute erosions distributed at villous tips was found in the anterior jejunum (Pl. II, Fig. 1). Absorptive cells at tips of villi and mucosal folds were often pleomorphous, hemispherically vaulted with changes in the microvillous area. Often release of oocysts from the epithelium was observed.

The following day post infection (6 DPI) sporadic erosions scarce distributed at

the tips of villi of the anterior jejunum and inflammatory infiltrate of the same character as at 4 DPI were observed. In enterocytes of the anterior jejunum stages of gametogony were present (Pl. I, Fig. 4) During examination of the inner surface of the intestine unapparent villous atrophy persisted and locally fusions of villi were observed. In contrast to 5 DPI, less epithelial erosions occurred at villous tips. In the duodenal mucosa release of *E. debliecki* oocysts was apparent (Pl. II, Fig. 2).

From 7 DPI to 11 DPI, in comparison with control animals, no differences were observed during histological examination nor during examination of the inner surface of the intestine.

Pathological changes observed in the intestine after infection with the coccidium *E. debliecki* were identical both at high and low doses. At high doses of oocysts ($4 \times 10^6$) pathological changes were observed already at 3 DPI and affected larger part of the small intestine. From 4 DPI to 5 DPI pathological changes reached after high doses of oocysts of *E. debliecki* the anterior jejunum, followed in the cranial direction to the duodenum and in the caudal direction to anterior parts of the middle jejunum.

**Transmission electron microscopy.** The ultrastructural study was concentrated on cellular changes of enterocytes in the region infected with the coccidium ***E. debliecki*** and on plasma cells in an inflammatory infiltrate in the lamina propria of the anterior jejunum from 4 DPI to 5 DPI. Cellular changes of enterocytes with developmental stages of *E. debliecki* were of various level. We observed nonsignificant cellular changes to necrobiosis of enterocytes in dependence on the age of the developmental stage of the coccidium.

Conspicuous changes in the microvillous zone were observed especially in enterocytes with mature stages of merogony or gametogony. Microvilli of these enterocytes were shortened, irregular, often forming groups of fused microvilli (Pl. III, Figs. 1, 2). Dilated mitochondria observed in the cytoplasm of infected enterocytes were localized particularly in apical part of enterocyte. Dilated vesicles of Golgi apparatus and free ribosomes were found supranuclearly. A great number of residual bodies was found in infected enterocytes (Pl. III, Fig. 3). The nuclei of enterocytes with the mature developmental stage of the coccidium *E. debliecki* was usually retracted into the apical part of enterocyte. In comparison with the nuclei of noninfected enterocytes, these nuclei of enterocytes were larger, spherical with smaller electron density of chromatin. Cellular changes were observed only in enterocytes with developmental stages of *E. debliecki*.

The cytoplasm of plasma cells of the inflammatory infiltrate contained abundant rough endoplasmic reticulum, Golgi apparatus and sporadic mitochondria (Pl. IV, Fig. 2). Cisternae of the rough endoplasmic reticulum of the plasma cells were often spherically dilated, filled with electron-dense, finely granulated material (Pl. IV, Fig. 3). Dilated cisternae of the rough endoplasmic reticulum were demarcated in some plasma cells with numerous ribosomes, in other cells endoplasmic reticulum was affected by necrobiosis and finely granular electron-dense material formed continuous strata.

### Results of parasitological examination

Prepatent period of all experimentally infected pigs neglecting the number of oocysts in an infective dose was from 5.5 to 6 days. Maximum number of *E. debliecki* oocysts was passed through the feces of the pigs from 6 DPI to 7 DPI. The patent period was 5 days; oocysts of *E. debliecki* were fully sporulated after incubation at 22 °C in 2.5 % pottassium dichromate solution after 9 days.



Examination of smears of the intestinal mucosa confirmed results of the histological examination. Developmental stages of *E. debliecki* were found in mucosal smears of the duodenum, anterior jejunum and proximal part of the middle jejunum.

## DISCUSSION

The results of Vetterling (1966b) who found out that the developmental cycle of *E. debliecki* takes place in the anterior jejunum, were confirmed in this paper. Similarly, to the results of Vetterling (1966b), it was detected that the extent of infection depends on the number of *E. debliecki* oocysts in an infective dose and that during the developmental cycle the displacement of the occurrence of developmental stages in the small intestine in the caudal direction can be observed. It was also found that at high doses of oocysts (4 millions of oocysts) the occurrence of developmental stages of *E. debliecki* together with its pathological changes is higher than in a lower number of oocysts in an infective dose (200 000 oocysts). Pathological changes even in large numbers of oocysts in an infective dose are restricted only to the epithelial barrier of upper parts of the mucosa where focal erosions of epithelial cells without larger necrosis of deeper layers of mucosa take place. Atrophy of the villi is slight during the infection with coccidia *E. debliecki* and no gross changes in the whole architecture of the intestinal mucosa take place. Moderate pathological changes of the intestinal mucosa did not influence the clinical state of experimentally infected pigs.

The developmental cycle of *E. debliecki* was observed in upper parts of mucosa of the anterior jejunum from 4 DPI to 5 DPI post infection with 4 millions of oocysts; developmental stages also occurred in the duodenum and anterior parts of the middle jejunum. Most abundant pathological changes occurred regularly in the anterior jejunum from 50 to 100 cm from the pylorus.

Cellular changes were found only in enterocytes infected with the developmental stage of *E. debliecki*. Changes in the microvillous zone were observed in infected enterocytes. Dilated mitochondria, free ribosomes and increased number of residual bodies were observed in the cytoplasm. Nuclei of infected enterocytes were spherical with smaller electron density than the nuclei of noninfected enterocytes. The degree of cellular changes of enterocytes dopended on maturity of the developmental stage of the coccidium. More apparent cellular changes were observed in enterocytes with mature developmental stages of *E. debliecki*. Ultrastructural changes of enterocytes post infection with the coccidium *E. nieschulzi* were described by Sheppard (1974). She mentions similar ultrastructural changes of enterocytes as we observed but she describes them in the area of the occurrence of developmental stages of the coccidium *E. nieschulzi* also in noninfected enterocytes.

An inflammatory infiltrate with conspicuously abundant eosinophils and plasma cells with Russell bodies was observed in the lamina propria during the whole period of the occurrence of pathological changes in the epithelium.

Russell bodies occurred in the form of numerous, spherical, often densely accumulated or locally fusing structures in the cytoplasm of plasma cells or in accumulations of spherical mulberry-shaped formations without nucleus or with a pyknotic nucleus near the periphery. Russell bodies were acidophilic in hematoxylin and eosin and were readily visible in azure eosin.

It was confirmed that electron-dense material in spherically dilated cisterns of the rugged endoplasmic reticulum of plasma cells forms a morphological basis of Russell bodies in a fine structure. Immunoglobulins were found in the content of

dilated cisterns of the rugged endoplasmic reticulum of plasma cells (Smith et al. 1972, Shiner 1983).

Visualization of immunoglobulin in a changed tissue is rare in the light microscopy. Eosinophilic material in the close vicinity of infectious agent called after the shape of peripheral structures asteroidal, actinomycetomatous or star-shaped bodies represents one of the expression of visualization of immunoglobulins in the histological picture. These are found during some mycotic infections of man and animals (Lurie 1963). Hoeppli (1932 — cit. Lurie 1963) described asteroidal material in the vicinity of bilharzial eggs. An evidence of the substance of these bodies during mycotic infections as the product of antigen-antibody reaction was given by Lurie and Still (1969), Prokš et al. (1972) and Müller et al. (1977).

Russell bodies represent a different form of the expression of local accumulation of immunoglobulin in inflammatory infiltrate in the region of pathogenic effect of the causative agent. In contrast to asteroidal bodies an apparent linkage on the cytoplasm of plasma cells was observed and the release of immunoglobulin from cisterns of the rugged endoplasmic reticulum takes place only after its large accumulation followed with necrobiosis of plasma cells.

Smith et al. (1972) mention that the cytoplasm of plasma cells in animals often contains hyalin spheres named Russell bodies. However, descriptions of Russell bodies in inflammatory infiltrate during particular infections of animals are given rarely. Dobberstein and Hemmert-Halswick (1928 — cit. Nieberle and Cohrs 1970) found Russell bodies in a tissue changed by inflammation during pernicious catarrhal bovine fever (coryza gangrenosa bovum). In Czechoslovakia, these were described in plasma cells of pseudomembranous inflammation during the dysentery of pigs (Vítovec and Vladík 1976).

Data from this study suggest that the coccidium *E. debliecki* is pathogenic for weaned pigs because it develops pathological changes in the anterior jejunum. The pathological changes are minute even at high infective doses ($4 \times 10^6$) and continuous damage of mucosal epithelial barrier does not occur and probably from these reasons the infection has a subclinical course. The conclusion of Lindsay et al. (1987) that *E. debliecki* does not cause diarrhea in weaned piglets, was confirmed.

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## ПАТОГЕННОСТЬ *EIMERIA DEBLIECKI* (DOUWES, 1921) И ПАТОЛОГИЯ НА УЛЬТРАСТРУКТУРНОМ УРОВНЕ У ЭКСПЕРИМЕНТАЛЬНО ЗАРАЖЕННЫХ СВИНЕЙ

Й. Витовец и Б. Коудела

**Резюме.** В настоящей работе показано, что после экспериментального заражения 24 поросят различными дозами ооцист *Eimeria debliecki* (200 000 и 4 миллиона) цикл развития *E. debliecki* протекает в передней части еюна, при высокой дозе ооцист также в двенадцатиперстной кишке и передней части среднего еюна. В зоне максимального скопления промежуточных стадий *E. debliecki* (от 50 до 100 см ниже пилорической части желудка) мы обнаружили патологические изменения, для которых характерна невыраженная атрофия крипт, редкие мелкие дефекты эпителия в верхних слоях слизистой и воспалительный ответ в собственной пластинке (lamina propria) передней части еюна. Воспалительный инфильтрат в lamina propria передней части еюна в период с 3-го по 5-й день после заражения (ДПЗ) содержал заметно высокое количество плазматических клеток с тельцами Рассела. Изменения клеток обнаружены только в энтероцитах содержащих стадии развития *E. debliecki*. В инфицированных энтероцитах обнаружены изменения в зоне микроворсинки, в цитоплазме энтероцитов обнаружены утолщенные митохондрии, свободные рибосомы и повышенное количество остаточных телец. Степень клеточных изменений энтероцитов коррелировал со зрелостью стадий жизненного цикла *E. debliecki*. На основе патологических изменений, обнаруженных с помощью световой и электронной микроскопии предполагаем, что *E. debliecki* является кокцидией патогенной для поросят, хотя у них не вызывает клинически выраженного заболевания.

## REFERENCES

ARNASTAUSKIENE T., 1985: Lietuvos naminiu ir laukiniu gyvuliu kokcidijos ir kokcidiozes. Vilnius "Mosklas".

BOCH J., PEZENBURG E., ROSENFELD, 1961: Ein Beitrag zur Kenntnis der Kokzidien der Schweine. Berl. Münch. Tierärztl. Wschr. 74: 449—451.

—, WIESENHÜTTER E., 1963: Über einzelne Stadien der endogenen Entwicklung des Schweinekokzids *Eimeria debliecki* Douwes, 1921. Berl. Münch. Tierärztl. Wschr. 74: 236—238.

HORVÁTH G., NEMES CS., 1988: A survey of swine coccidiosis in Hungary. In: Abstracts of the Vth European Multicolloquium of Parasitology. Edited by T. Kassai, Budapest, Hungary, p. 133.

KARNOVSKY M. J., 1965: A formaldehyde-paraformaldehyde fixative of high osmolarity for use in electron microscopy. J. Cell. Biol. 27: 137a—138a.

KUTZER E., 1960: Über die Kokzidien des Schweines in Österreich. Z. f. Parasitenkunde 19: 541—547.

LINDSAY D. S., ERNST J. V., CURRENT W. L., STUART B. P., STEWART T. B., 1984: Prevalence of oocysts of *Isospora suis* and *Eimeria* spp. from sows on farms with and without a history of neonatal coccidiosis. J. Am. Vet. Med. Assoc. 185: 419 to 421.

—, BLAGBURN B. L., BOOSINGER T. R., 1987: Experimental *Eimeria debliecki* infections in nursing and weaned pigs. Vet. Parasitol. 25: 39—45.

LURIE H. I., 1963: Histopathology of sporotrichosis. Notes on the nature of the asteroid body. Arch. Path. 75: 421—437.

—, STILL W. J. S., 1969: The "capsule" of *Sporotrichum schenckii* and the evolution of the asteroid body. Sabouraudia 7: 65—70.

MÜLLER J., TAKAMIYA H., JAEGER R., 1977: Elektronmikroskopische Darstellung von Immunreaktionen an *Candida*-Zellen: asteroid bodies bei *Candida albicans* im Urin von Nephritis-Patienten. Sabouraudia 15: 87—93.

NIEBERLE N., COHRS P., 1970: Lehrbuch der speziellen pathologischen Anatomie der Haustiere. VEB Gustav Fischer Verlag, Jena, 372 pp.

PROKŠ C., VITOVEC J., VLADÍK P., 1972: Asteroide Körperchen bei Aspergillose. Mykosen 15: 427—430.

ROMERO J. R., LICZANO J. H., 1971: Revision de los generos: *Eimeria* e *Isospora* (Protozoa, Eimeridae) del *Sus scrofa domestica* (L). Descripciones de las *Eimeria guevarai* e *Isospora neyrai* ns. spp. Rev. Iber. Parasitol. 31: 2—13.

SHEPPARD A. M., 1974: Ultrastructural pathology of coccidian infection. J. Parasitol. 60: 369—371.

SHINER M., 1983: Ultrastructure of the small intestinal mucosa. Springer-Verlag; Berlin, Heidelberg, New York, pp. 41—42.

SMITH H. A., JONES T. C., HUNT R. D., 1972: Veterinary Pathology, Lea Febiger, Philadelphia, pp. 178—179.

VARGHESE T., 1986: Porcine coccidia in Papua New Guinea. Vet. Parasitol. 21: 11—20.

VETTERLING J. M., 1966a: Prevalence of coccidia in swine from six localities in the United States. Cornell Vet. 56: 155—156.

—, 1966b: Endogenous cycle of swine coccidium *Eimeria debliecki* Douwes, 1921. J. Protozool. 13: 290—300.

VÍTOVEC J., VLADÍK P., 1976: Colitis cystica profunda in pseudomembranous inflammation during dysentery of pigs. Vet. Med. (Prague). 21: 475—481.

WARMKE H. E., SHEU-LING J. L., 1976: Improved staining procedures for semithin epoxy sections of plant tissues. Stain Technol. 51: 179—185.

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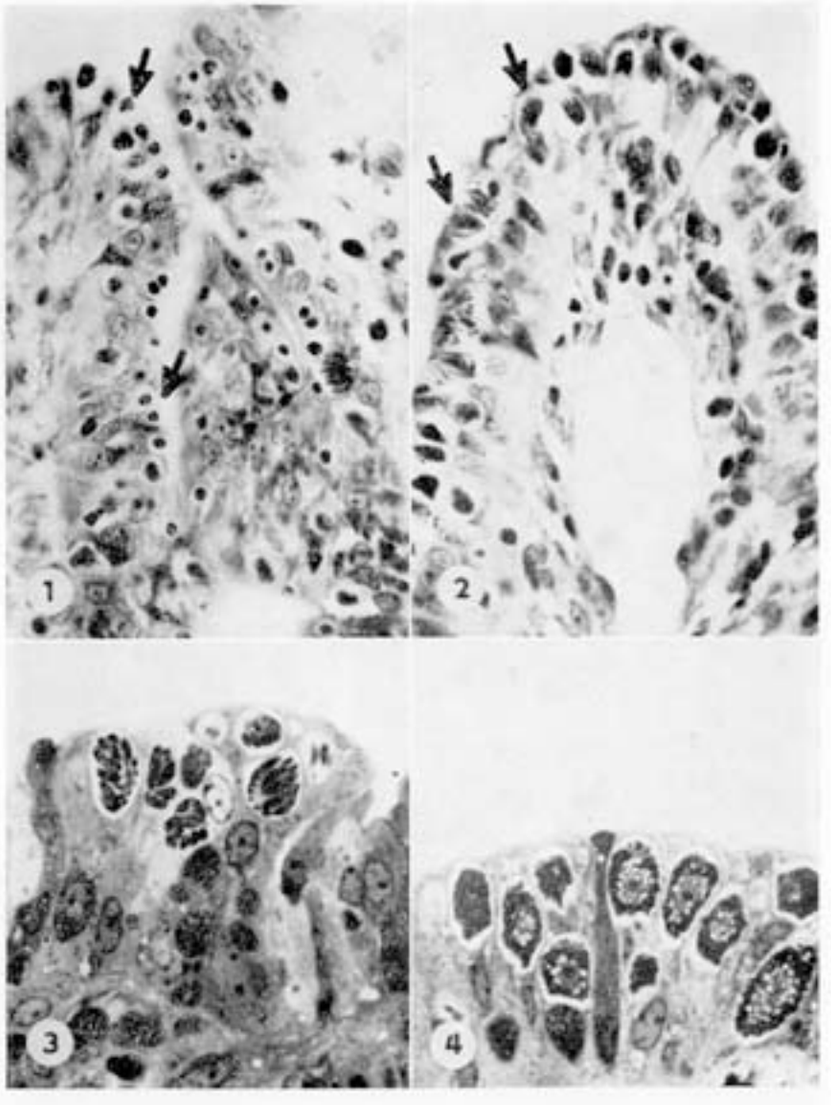



**Fig. 1.** Immature meronts of *E. debliecki* in enterocytes of villous tips of the anterior jejunum (arrows) — 3 DPI (HE, 320×). **Fig. 2.** Mature meronts of *E. debliecki* in enterocytes of villous tips of the anterior jejunum (arrows) — 4 DPI (HE, 320×). **Fig. 3.** Mature meronts of *E. debliecki* in enterocytes of the anterior jejunum — 4 DPI (semithin section, polychrome staining according to Warmke, 450×). **Fig. 4.** Stages of gametogony of *E. debliecki* in enterocytes of the anterior jejunum — 6 DPI (semithin section, polychrome staining according to Warmke, 500×).



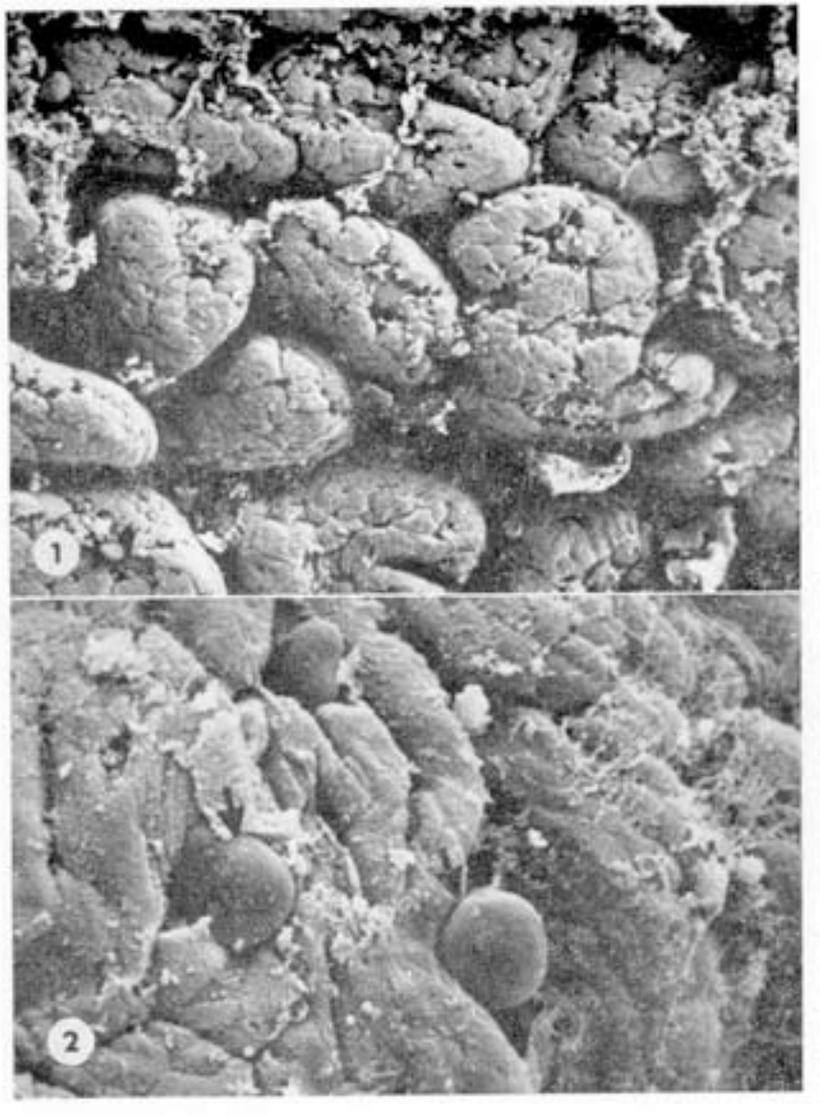



**Fig. 1.** Epithelial erosions of atrophied villi of the anterior jejunum — 5 DPI (SEM, 325×). **Fig. 2.** Shedding of *E. debliecki* oocysts from the duodenal mucosa (arrows) — 6 DPI (SEM, 2300×).

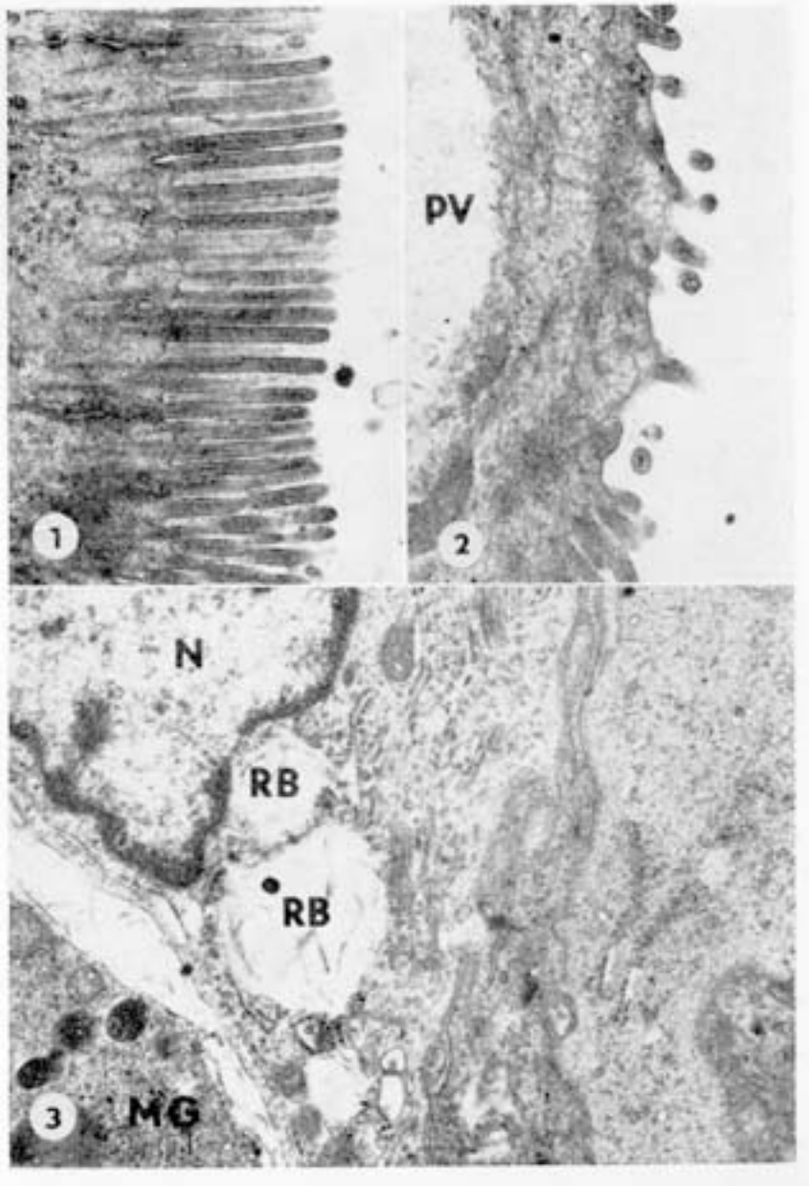
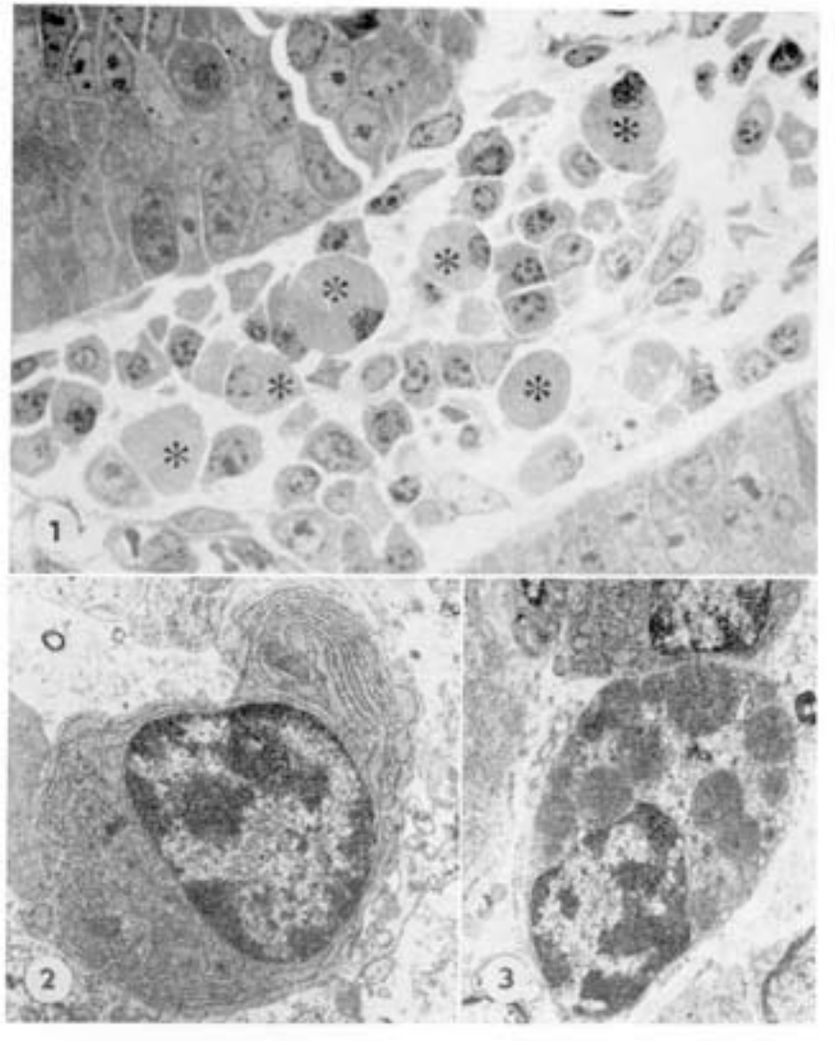



**Fig. 1.** Regular microvilli of enterocyte with no developmental stage of *E. debliecki*, the anterior jejunum — 5 DPI (TEM, 20,000×). **Fig. 2.** Shortened and irregular microvilli of enterocyte with mature macrogamont of *E. debliecki*, the anterior jejunum — 5 DPI, PV — parasitophorous vacuole (TEM, 20,000×). **Fig. 3.** Perinuclear area of enterocyte with macrogamont of *E. debliecki*, the anterior jejunum — 5 DPI, RB — residual bodies, MG — macrogamont of *E. debliecki*, N — nucleus of enterocyte (TEM, 20,000×)






**ig. 1.** A great number of plasma cells with Russell bodies (asterisk) in the propria of villi of the nterior jejunum — 4 DPI (semithin section, polychrome staining according to Warmke, 1,250×). **ig. 2.** Mature plasma cell with parallel cisterns which contain ribosomes RER, the lamina propria f the anterior jejunum — 4 DPI (TEM, 11,750×). **Fig. 3.** Plasma cell with cisterns which contain ussell bodies, the lamina propria of the anterior jejunum — 4 DPI (TEM, 11,750×).