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# *Schellackia ptyodactyli* sp. n. of the fan-footed gecko *Ptyodactylus hasselquistii* from the rift escarpment of the lower Jordan Valley

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**Abstract.** *Schellackia ptyodactyli* sp. n. is described from the fan-footed gecko *Ptyodactylus hasselquistii* (Donndorf) found the lower Jordan Valley, Cis-Jordan. Endogenous development was studied in geckoes necropsied 7–11 days after being inoculated with blood containing sporozoites from naturally infected geckoes of the same species. Merogony and gamogony/oogony stages, as well as sporozoites, are described by light and electron microscopy. Merogony stages, microgamonts and sporozoites conformed in fine structure to that of other eimerian coccidia, whereas wall forming bodies of the macrogamonts showed some divergence from the general pattern characteristic of eimerians and *Schellackia* cf. *agamae*. Merogony stages occurred simultaneously with gamonts and sporozoites. In the blood, sporozoites entered leucocytes, thrombocytes and erythrocytes. Parasitaemia persisted for up to 2 years in some naturally infected geckoes in captivity.

Examination of blood smears taken from fan-footed geckoes *Ptyodactylus hasselquistii* (Donndorf, 1789) (Geckonidae: Lacertilia), caught on the rift escarpment of the lower Jordan Valley, revealed leukocytic and erythrocytic infection with *Schellackia*-like sporozoites. Experimental infection of uninfected fan-footed geckoes confirmed the identity of these sporozoites as a previously unknown species of *Schellackia* – *S. ptyodactyli* sp. n. It is the first record of a member of this genus in geckoes. In this communication, this new species is described by light and electron microscopy, and differentiated from the other known species of *Schellackia*.

## MATERIALS AND METHODS

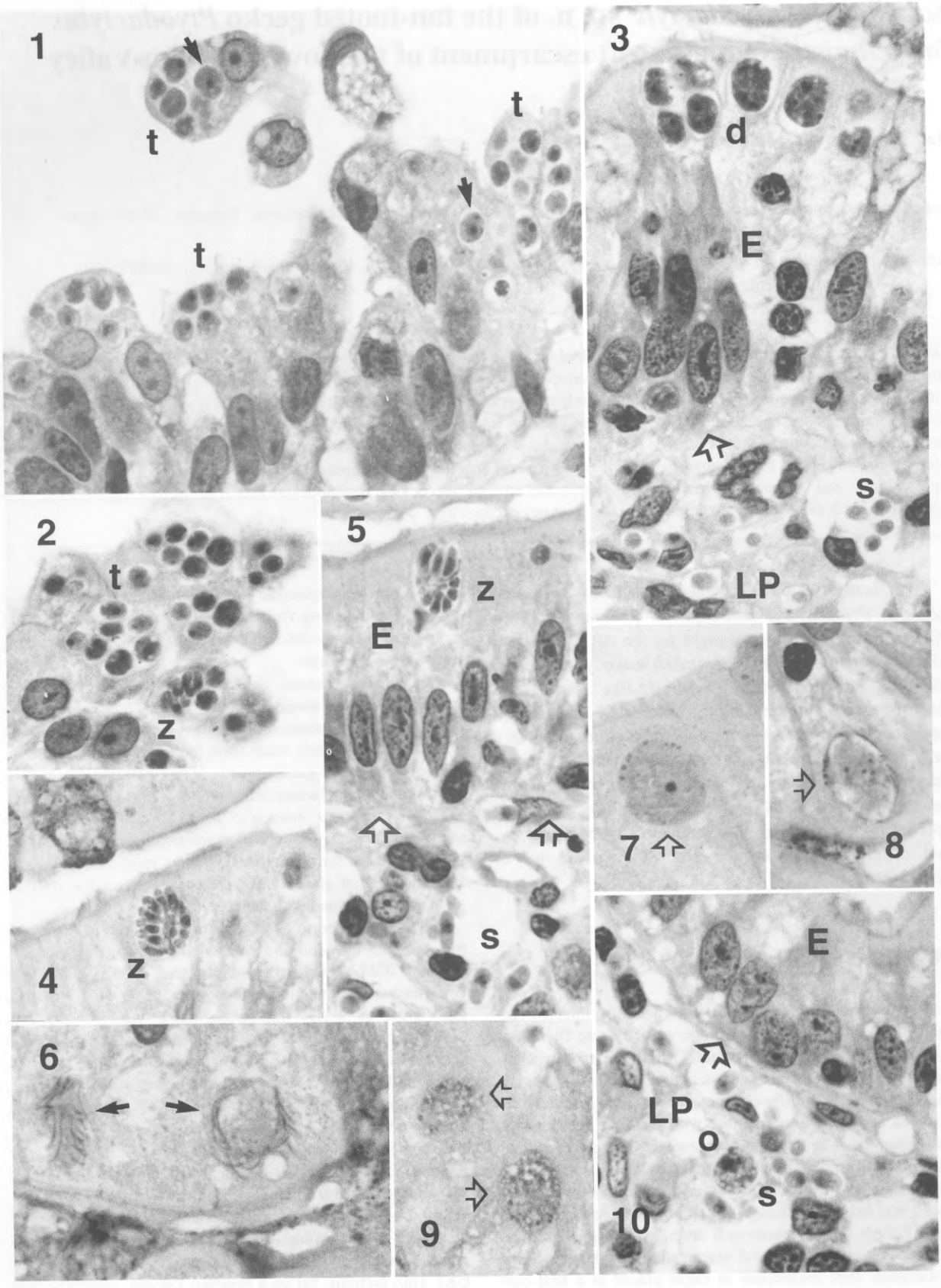
Blood containing sporozoites from naturally infected fan-footed geckoes was injected intraperitoneally into five uninfected geckoes of the same species. Of these, four were sacrificed 7, 8, 10 and 11 days later for parasitological examination. An additional four geckoes were similarly inoculated and kept alive together with the fifth gecko of the first group to monitor the appearance of sporozoites in the blood cells. The course of parasitaemia was also monitored in two naturally infected geckoes. An additional uninfected gecko was fed on the shed tail of an infected gecko and sacrificed 8 days later. Experimental animals, timing of inoculations and necropsies, or blood examinations are listed in Table 1. Geckoes were maintained in cages placed in a heat-controlled room, at ambient temperatures of 25–29°C, and fed flour-beetle larvae.

Freedom from infection in the experimental animals was ensured by collecting them when they were very young and monitoring them for infection by repeated blood examinations for a further 2 months.

In sacrificed geckoes, the location of the infection in the intestine was determined by direct microscopic examination of fresh squash preparations under a coverslip, and from Giemsa-stained smears made from the various parts of the intestine. Parts of the gut found to contain parasites were taken for histology and electron microscopic study. Smears and histological specimens were also prepared from livers of the sacrificed geckoes. Blood was examined at 3-days to 1-week intervals. Blood for smears was obtained from a clipped clow. Blood smears on glass slides were air dried, fixed in absolute methanol and stained with Giemsa.

For histology portions of the gut were fixed in 10% neutral, buffered formalin and embedded in glycol methacrylate medium (GMA medium of Agar Co., UK). Sections of GMA-embedded material were cut at 3.0 to 4.0 μm with a glass knife on a JB4 microtome. Some were stained with Meyer's haemalum and eosin; others were post-fixed in aqueous Bouin's fluid for 20 min., washed in 70 % ethyl alcohol until colourless, stained in Giemsa as were the smears, and differentiated in 70–30 %, 30–70 %, 0–100 % acetone-xylol mixtures.

For electron microscopy, portions of the intestine were fixed in 2.5 % glutaraldehyde in cacodylate buffer (0.1 M, pH 7.4) for 24 h at 4.0°C, rinsed repeatedly in the same buffer, post-fixed in 1.0 % osmium tetroxide in the same buffer for 1 hour and, after rinsing in the same buffer, dehydrated in graded alcohols and embedded in Agar 100® resin (Agar Co., UK). Thin sections, cut on a Reichert Ultracut ultratome with a diamond knife, were stained on grids with uranyl acetate and lead citrate and examined in a Jeol 100CX TEM.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## RESULTS

We propose assigning the below described *Schellackia* to a new species:

### *Schellackia ptyodactyli* sp. n.

Type host: *Ptyodactylus hasselquistii* (Donndorf, 1789).
Type locality: Lower Jordan Valley (20 km north of Jericho), Cis-Jordan.

### Experimental infections

Five of the six sacrificed geckoes inoculated with sporozoite-containing blood became infected; the one fed the infected gecko tail remained uninfected. Of the five geckoes inoculated with infected blood and reserved to follow up parasitaemia, sporozoites failed to appear in one (Table 1). Endogenous epithelial stages were located mainly in the anterior intestine – the duodenum and anterior part of the jejunum. Sexual stages occurred only in most anterior part of the duodenum, immediately adjacent to the pyloric region.

No correlation could be demonstrated in the experimentally infected geckoes beween the recovered range of endogenous developmental stages and the timing of the necropsies (Table 2). The gecko sacrificed on day 7 post infection (p.i.) was already with mature microgamonts, oocysts and numerous sporozoites in the lamina propria. Only young meronts were present in the gecko sacrificed 8 days p.i., and only meronts, merozoites and very young macrogamonts were found in the gecko sacrificed on day 11. Mature macrogamonts were found only in geckoes sacrificed on day 10 p.i.; these coexisted with sporozoites in both the intestinal and extraintestinal tissue.

**Light microscopy.** Merogonous development took place at the apical end of the gut epithelial cell. In particularly heavy infections, epithelial cells were invaded by numerous young meronts and gamonts measuring 2–4 × 1.6–3.5 μm (Fig. 1). In some cells these young zoites were accompanied by a few dividing meronts and even late ones which were already differentiating into merozoites (Fig. 2). In histological sections these cells, with up to 13 zoites, were grossly hypertrophic, displaced onto the mucosal surface and ultimately shed, degenerated, into the gut lumen (Figs. 1, 2). This hypertrophy, however, did not involve the host-cell nucleus. Rounded, mature meronts of 7–8 × 6–7 μm (Fig. 3) yielded up to 36 merozoites by ectomerogony, which formed around a large residuum (Figs. 4, 5). Microgamonts, reaching 9–12 × 7–10 μm in size at maturity, were established in the basal layer of the epithelium (Fig. 6). Mature macrogamonts of 11–12 × 7–10 μm, in size, were recognized by their nucleus containing a central, round nucleolus. They contained the presumed wall-forming organelles: very distinct violet-staining granules (Figs. 7, 8) and faintly eosinophilic round vesicles, some of which contained a dense violet granule, and a variable number of amylopectin granules (Fig. 11a). Zygotes and young oocysts containing many amylopectin granules still occurred in the epithelial layer

**Table 1.** List of attempted infections, examined geckoes and timings of necropsies and blood examinations (d.p.i. – days post infection, d.p.c. – days after capture).

| Source of inf. | How infected | No. | Sacrificed d.p.i. | Only checked blood | Post./Neg. |
|---|---|---|---|---|---|
| Gecko A Nat. infection | Inoculated blood | G1 | 10 d.p.i. | – | Pos. |
| Gecko B Nat. infection | -do- | G2-5 | 7, 8, 10, 11 d.p.i. | – | Pos. |
| Gecko B | -do- | G6 | – | 21, 30 d.p.i. | Neg. |
| Gecko B | Fed on tail. | G7 | 8 d.p.i. | – | Neg. |
| | Natural | G8 | – | 0 – 69 d.p.c. | Pos. |
| | Natural | G9 | – | 0 – 44 d.p.c. | Pos. |
| G8 | Inoculated blood | G10–13 | – | 21– 34 d.p.i. | Pos. |

← **Figs. 1–10.** Light microscopic view of endogenous stages of *Schellackia ptyodactyli* seen in histological sections stain with Meyer's haemalum–eosin. **Fig. 1.** Gut epithelial cells massively infected with juvenile meronts (t) and gamonts (arrows), resulting in cell hypertrophy and sloughing (× 1200). **Fig. 2.** Same as Fig. 1, showing dividing meronts (z) among the juvenile stages (t) (× 1300). **Fig. 3.** Differentiating meronts (d) occurring in the gut epithelial cells (E) concurrently with sporozoites (s) in the *lamina propria* (LP, open arrow: basal lamina)(× 1300). **Fig. 4.** Merozoites (z) differentiating around the meront's residual body (× 1200). **Fig. 5.** Dividing meronts (z) in the epithelium (E) co-existing with sporozoites in the *lamina propria* below the basal lamina (open arrow) (× 1300). **Fig. 6.** Two mature microgamonts at the base of a gut epithelial cell (× 1272). **Fig. 7.** Macrogamont in the gut epithelium showing large nucleolus and violet granules (× 1454). **Fig. 8.** Late stage macrogamont in the gut epithelium showing violet granules and amylopectin granules (× 1565). **Fig. 9.** Zygotes or early oocysts in the gut epithelium with many amylopectin granules and wall-forming granules (× 1500). **Fig. 10.** Oocysts (o) and sporozoites (s) in the *lamina propria* (LP, E: gut epithelium, open arrow: basal lamina) (× 1100).

**Table 2.** Developmental stages encountered in smears, sections and ultrathin sections of the intestines of fan-footed gecko inoculated with blood sporozoites.

| D.p.i. | Meronts | Merozoites | Young gamonts | Mature microgamonts | Mature macrogamonts | Oocytes | Sporozoites |
|---|---|---|---|---|---|---|---|
| 7 | | | | + E | | + E,L | +++ L |
| 8 | ++++ E | | | | | | |
| 10 | | | + E | + E | +++ E | + E | |
| 10 | ++++ E | +++ E | | | + E | + E,L | ++++V |
| 11 | +++++ E | +++ E | +++ E | | | | |

Location of infection: E – gut epithelium, L – *lamina propria*, V – all viscera.

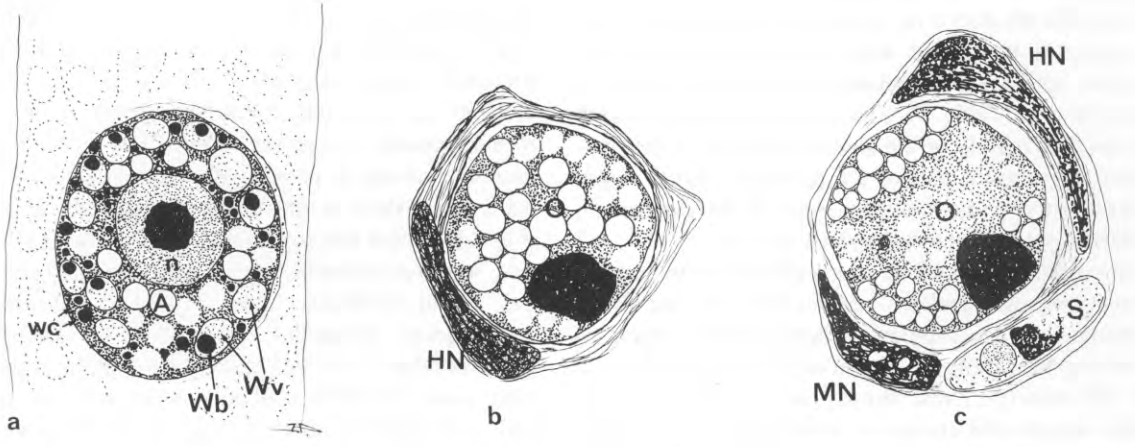
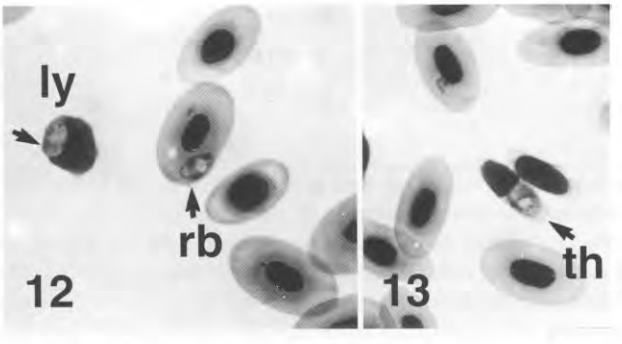
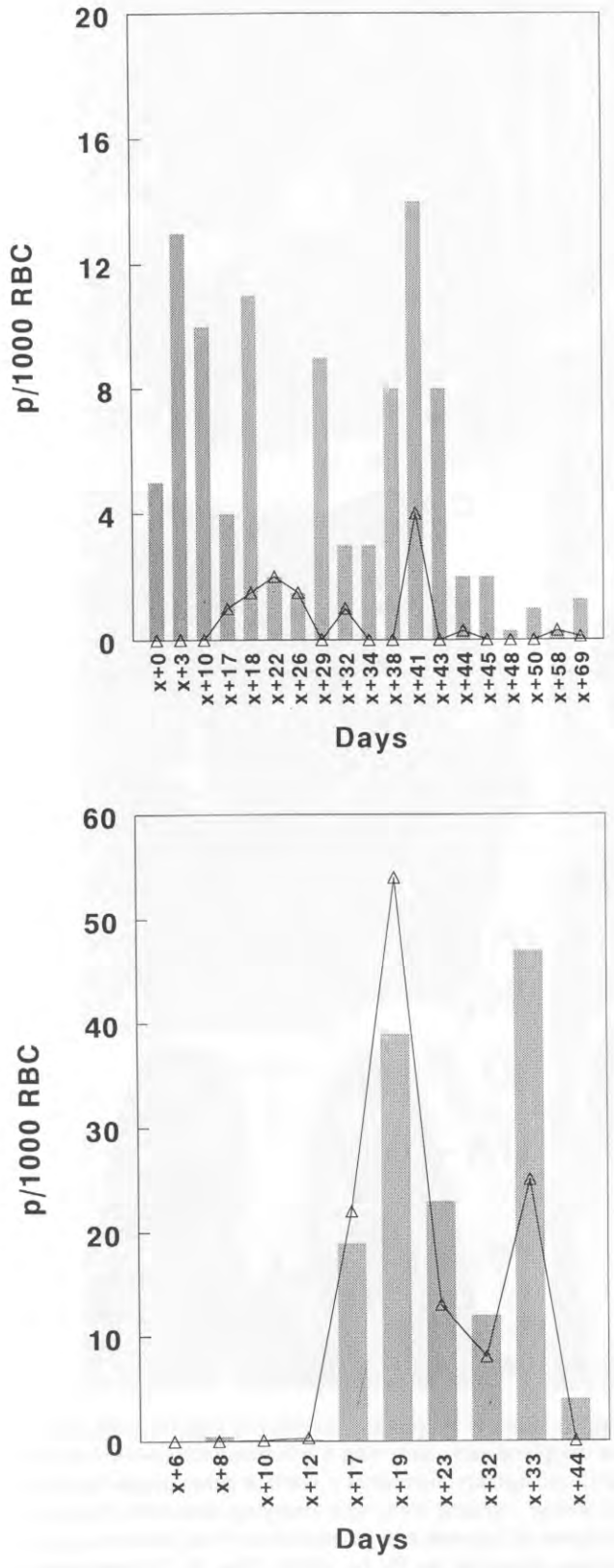



**Fig. 11.** Line drawings from HE-stained histological section (× 5000). **a** – mature macrogamont in the gut epithelium (A, amylopectin granules; n, nucleus; Wb, large violet granules inside Wv – an eosinophilic vesicle (~WFB 2); wc, small violet granules (~WFB 1). **b** – Oocyst (O) in the *lamina propria* enclosed within a shrunken host cell (HN = host-cell nucleus). **c** – Oocyst (O) enclosed together with its host cell (HN = host-cell nucleus) inside a presumed macrophage (MN = macrophage nucleus) which also hosts a sporozoite (S).




**Figs. 12, 13.** Sporozoites inside a lymphocyte (ly), an erythrocyte (rb) and thrombocytes (th) (blood smear, Giemsa-stained, × 1200).

(Fig. 9). Non-sporulated oocysts occurred more frequently in the lamina propria than they did in the epithelial layer. They were enclosed in a solid wall and were usually located within a widened inclusion, limited by an outer envelope (Fig. 10). The oocyst's smaller dimensions (7–10 μm in diameter), as compared to 11–12 μm for the macrogamont, appeared to result from shrinkage during processing, possibly due to the former's greater resistance to the entry of fixatives or embedding media. In the *lamina propria* oocysts were seen to be within an inclusion contained within what appeared to be the host cell residue – a fine cytoplasmic layer which sometimes also contained a large nucleus (Fig. 11b). Such oocysts, together with their presumed host cell residues were seen within macrophages. In




**Figs. 14, 15.** Course of parasitemia in two naturally, high and low infected captive geckoes (histogram: ‰ infected erythrocytes, line: no. infected leucocytes per 1000 erythrocytes).

addition to the oocyst some of macrophages also contained sporozoites (Fig. 11c).

**Sporozoites** of 4.0–4.6 × 1.0–1.3 µm, in the lamina propria, were either extracellular or contained within cells, some of which were macrophages. They occurred concurrently with young and dividing meronts in the mucosal epithelium (Figs. 3, 5, 10). In the liver only few sporozoites were seen, but they were not within hepatocytes; in the lungs and the spleen, most of them were within blood cells in the sinuses. None were found in the melano-macrophages. In the circulating blood sporozoites occurred in erythrocytes, thrombocytes, granulocytes and lymphocytes (Figs. 12, 13).

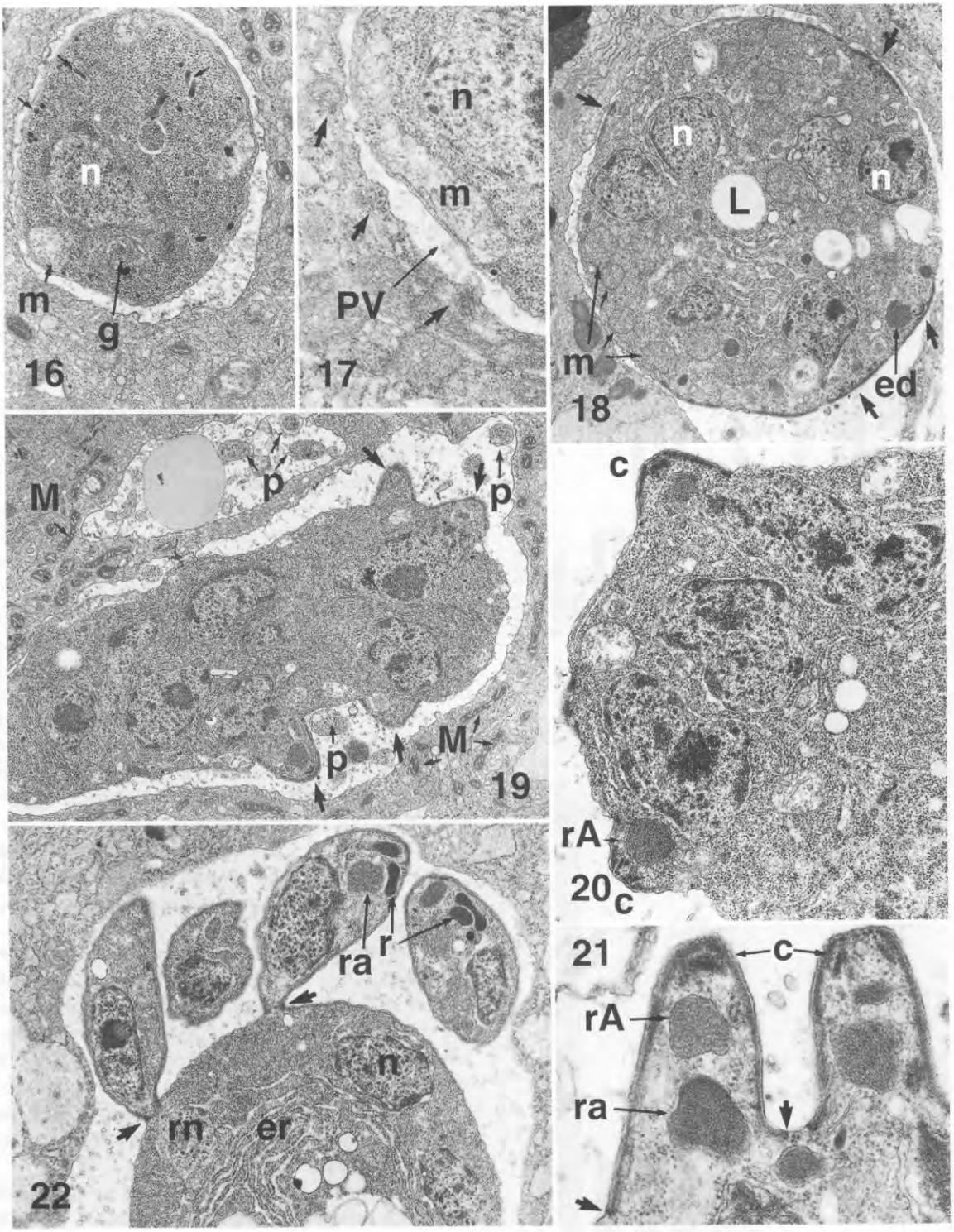
**Natural infections and the course of parasitemia**

The prevalence of natural infections in gecko populations (randomly collected, in different seasons, and including juveniles, subadults and adults), ranged from 5 to 58 % in one location on the escarpment and 13 to 38 % in another location (unpublished data). The levels of parasitemia in natural and inoculated infections was variable, with less than 0.1 to 5 % of the blood cells infected. It never exceeded this level even in inoculated infections. At the early phase of parasitemia, sporozoites were common in the non-erythrocytic cells, but later they occurred mainly in the erythrocytes. The natural infection was followed in captive lizards for up to 69 days (Figs. 14, 15); data from other fan-footed geckoes held in captivity indicated the persistence of a very low-level sporozoite blood infection (< 2 ‰) for over a year.

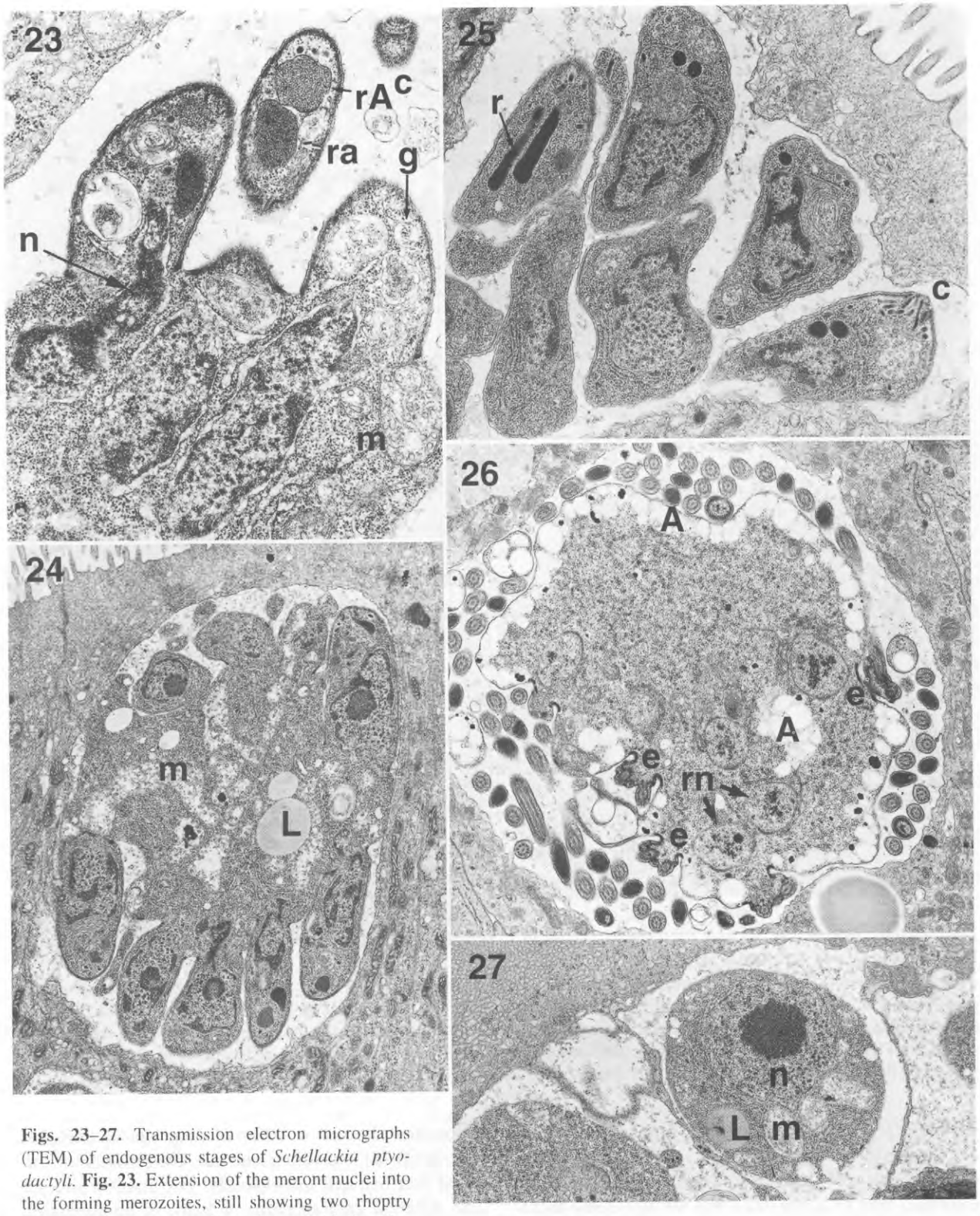
Geckoes in the same habitats were infected with mites, both *Geckobiella* sp. and larval Trombiculidae, but in neither was it possible to demonstrate the presence of sporozoites.

**Electron microscopy**. Juvenile stages invading gut epithelial cells either still retained a merozoite-type pellicle or were already bound by a single membrane (Fig. 16). The cytoplasm of these juvenile forms revealed one or several large mitochondria, a lipid vacuole, and a few micronemes. The parasitophorous vacuole (PV) contained globular debris, while at its boundary there were several small protrusions into which a small mitochondrion from the host cytoplasm extended (Fig. 17).

The developing meronts contained expanded nuclei, suggesting their division via fragmentation. Following nuclear division in the meronts, electron-dense plaques were formed below the cell-limiting membranes and marked the sites where merozoite primordia would emerge (Fig. 18). Merozoites were formed by exogenesis (Figs. 19–24). Subsequent to plaque formation, apical complex conoids of the emerging merozoite appeared, accompanied beneath by a large rhoptry anlagen (Figs. 19, 20). The meront nuclei elongated and extended into the forming merozoites (Fig. 23). Merozoite primordia also developed along deep invaginations

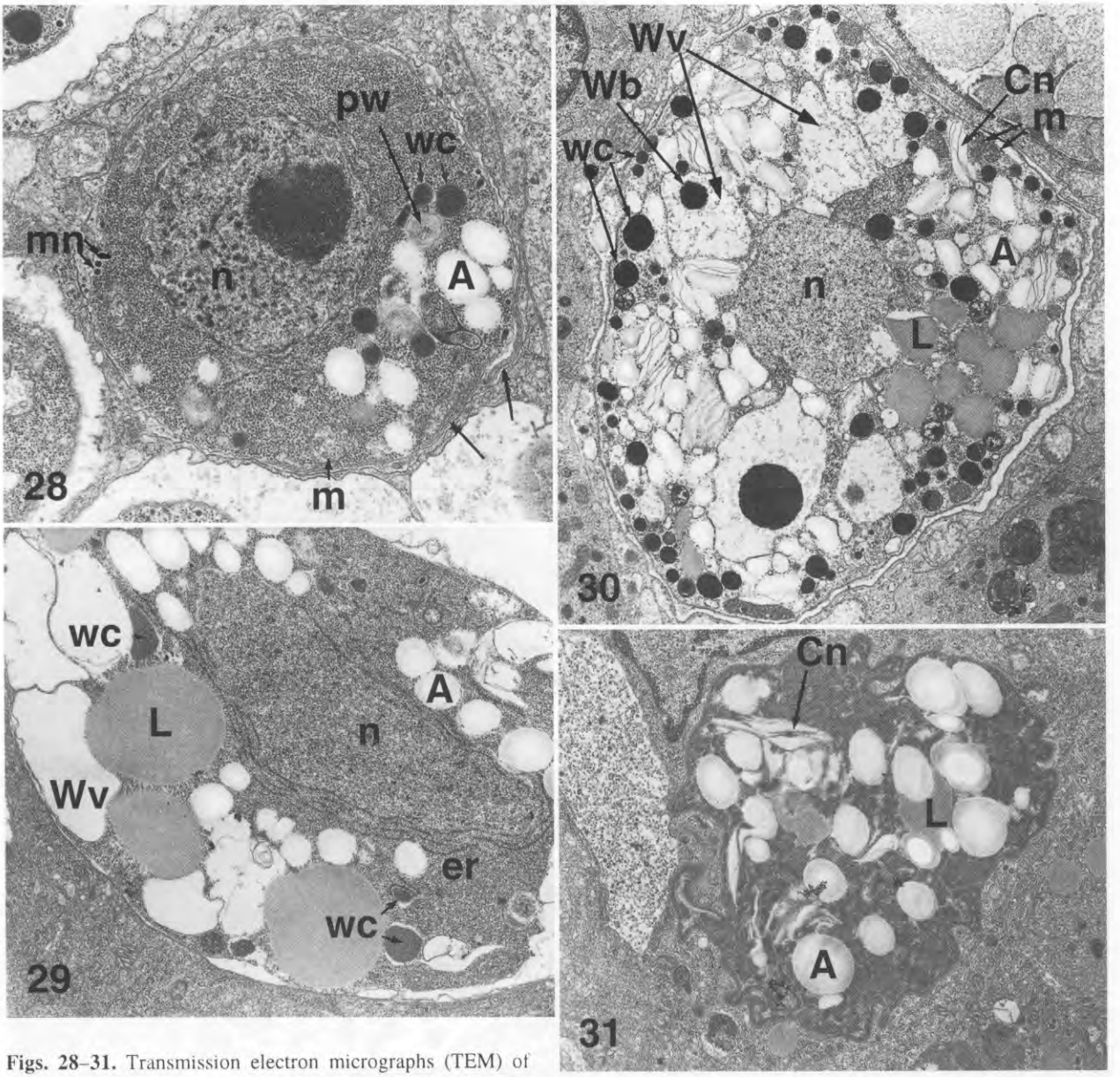



**Figs. 16–22.** Transmission electron micrographs (TEM) of endogenous stages of *Schellackia ptyodactyli*. **Fig. 16.** Young meront (arrows: micronemes) (× 16900). **Fig. 17.** Protrusions of host cytoplasm each containing a mitochondrion (arrows) at the border of the PV (× 24900). **Fig. 18.** Meronts with divided nuclei, rudimentary merozoites – electron dense plaques and an emerging conoid (bold arrows) (× 9900). **Fig. 19.** Differentiating meront – general view, with emerging merozoites (bold arrows). Membrane-bound inclusions of ribosome-rich parasite cytoplasm (p) appearing to be protrusions from the meront surface. Many mitochondria (M) aggregating in the host cell cytoplasm alongside the PV (× 10000). **Fig. 20.** Differentiating meront with emerging merozoites showing an already organized apical complex and rhoprty anlagen (rA). Nuclei expanding into the base of the merozoite (× 16200). **Fig. 21.** Emerging merozoites with apical complexes, two rhoptry anlagen, and a "ring" which is formed at the junction between the merozoite pellicle and the meront plasmalemma (arrows) (× 35750). **Fig. 22.** Late stage differentiating merozoite with rhoptries (r) alongside rhoptry anlagen (ra), with a juncture ring (bold arrow). Meront residuum contains active (n) and residual nuclei (rn) and extensive ER (× 10750). Abbreviations – see p. 167.




**Figs. 23–27.** Transmission electron micrographs (TEM) of endogenous stages of *Schellackia ptyodactyli*. **Fig. 23.** Extension of the meront nuclei into the forming merozoites, still showing two rhoptry anlagen, c-section revealing the conoid tip of a merozoite (× 19500). **Fig 24.** Meront in an advanced stage of division into merozoites; many large mitochondria (m) filling the meront residuum (× 7342). **Fig. 25.** Free merozoites (× 15770). **Fig. 26.** Mature microgamont with peripherally arranged microgametes (e). The cytoplasm contains residual nuclei (rn) (× 8840). **Fig. 27.** Early macrogamont (× 8250).

**Abbreviations** to Figs. 16–35: **A** – amylopectin granules, **c** – conoid, **Cn** – canaliculi, **er** – endoplasmic reticulum, **g** – golgi apparatus, L – lipid vacuole, **m** – mitochondria, **mn** – micronemes, **n** – nucleus, **PV**– parasitophorous vacuole, **R** – refractile body, **r** – rhoptries, **rA** – rhoptry anlagen (granular), **ra** – rhoptry anlagen (dense).




**Figs. 28–31.** Transmission electron micrographs (TEM) of endogenous stages of *Schellackia ptyodactyli*. **Fig. 28.** Premature macrogamont, still with a few micronemes (mn), containing amylopectin granules, wall-forming bodies (pw) and their presumed precursors (pw): arrows point to the multimembranous cell wall (× 14900). **Fig. 29.** Presumed mature macrogamont showing type-1 wall-forming body (wc) and large vesicles which appear to enclose the type-2 wall-forming body (Wv) (× 10840). **Fig. 30.** A zygote with numerous, variable-sized type-1 wall-forming bodies (wc) and vesicles (Wv) which contain the large type-2 wall-forming bodies (× 7060). **Fig. 31.** Defunct (?) young oocyst (× 9850). Abbreviations – see p. 167.

in the surface of some meronts. The differentiating meront's cytoplasm contained many mitochondria, extensive endoplasmic reticulum (ER) accompanied by cisternae, lipid vacuoles and a few electron-dense inclusions of unknown nature (Figs. 18–20). In the PV, membrane-bound bodies densely filled with ribosomes, seemed to be pseudopodium-like extentions of the meronts' surface (Fig. 19). An electron-dense ring marked the border between the meront wall and the consolidating merozoite pellicle (Figs. 21, 22). The emerging merozoites showed a differentiating apical complex with details of the future conoid. The primordia contained first one and subsequently two rhoptry anlagen with different grain densities, a frame of microtubules, mitochondria and ER (Figs. 20, 21, 23). In more differentiated merozoites, electron-dense differentiated rhoptries occurred alongside still undifferentiated rhoptry anlagen (Fig. 22). The cytoplasm of the meront residuum contained very large mitochondria, residual nuclei and an extensive concentric network of ER (Figs.

22, 24). Formed progeny of merozoites revealed nuclei with either marginally distributed chromatin, i.e. merogony or microgamont progeny (Fig. 25) or centrally aggregated chromatin – i.e. macrogamont progeny.

Mature microgamonts with differentiated microgametes (Fig. 26) contained a fairly homogeneous cytoplasm with a few to numerous residual nuclei entangled in ER, a few small mitochondria and amylopectin granules aligned beneath the cell boundary where there were no differentiating microgametes.

Juvenile stages of future macrogamonts had a nucleus with a prominent central nucleolus. Their cell boundary either still retained a pellicle or has already been reduced to a single unit membrane. The cytoplasm contained large and small mitochondria and one large lipid vacuole, and still retained a fair number of micronemes (Fig. 27). Later-stage macrogamonts contained some forming amylopectin granules and electron-dense type-1 wall-forming bodies (WFB). Some membrane-enclosed bodies of medium density may have represented precursors of WFB. The single-unit cell boundary was accompanied beneath, in parts, by an additional layer of two to three closely applied membranes. There were many peripherally located mitochondia, and a few scattered micronemes (Fig. 28).

In mature macrogamonts, presumably after fertilization (Figs. 29, 30), the nucleolus was absent from the nucleus in the plane of the sections examined. The wall consisted of a single unit-membrane. The cytoplasm contained bean-shaped amylopectin granules, canaliculi, a fair number of lipid vacuoles and variable-sized vesicles with flocculent material. Some contained a very prominent, large electron-dense body, presumably a type-2 WFB, and numerous small and medium size electron-dense bodies – the presumed type-1 WFB. In the macrogamont shown in Fig. 29, there were few dense bodies, but many may have been extracted during the embedding proceduce. The cytoplasm also contained a network of ER, which was organized into concentric whorls around the nucleus (Fig. 29). Some of the zygotes and young oocysts were in a state of disintegration (Fig. 31). All zygotes were contained within functional host cells, although in some, altered cytoplasmic organelles (shrunken mitochondria) and excessive vacuolation suggested the onset of a degenerative processes.

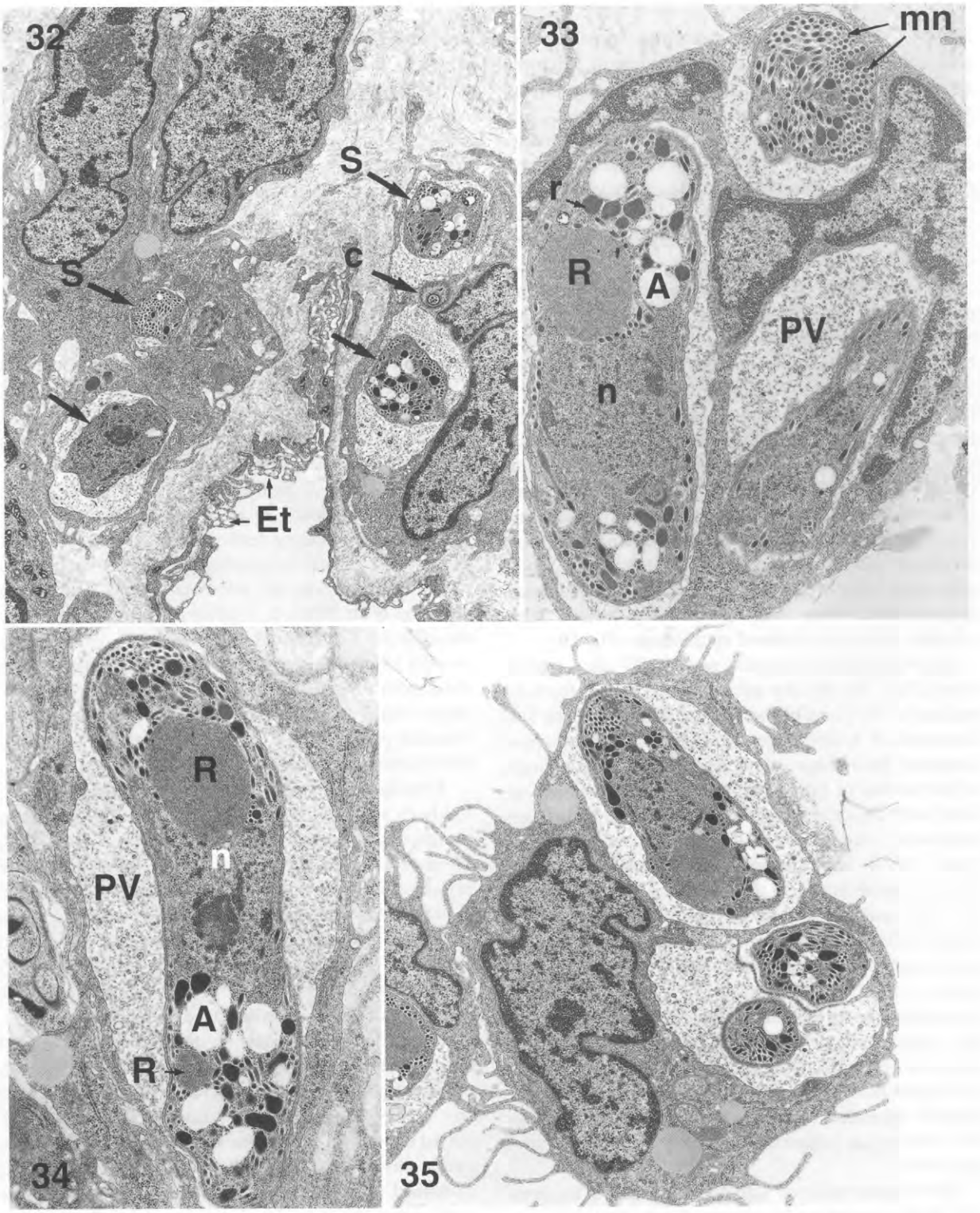
Sporozoites occurred within various cells of the *lamina propria*, including leucocytes. Single host cells contained three or more parasites (Fig. 32). Each sporozoite was located inside an individual expanded PV, filled with granules and membranous debris, and the PV boundries were intact or only slightly fragmented (Figs. 33–35). Exceptionally, sporozoites, or their conoid part (Fig. 32), were seen extending outside the PV borders. Sporozoites were readily recognized by their two large refractile bodies and numerous micronemes in their anterior as well as posterior ends. Rhoptries extended to the posterior end (Figs. 33, 34). Sporozoites contained variable numbers of amylopectin granules (Figs. 33–35). Host cells did not show any detectable structural damage.

## DISCUSSION

Although species of *Schellackia* show only limited interspecific morphological diversity, they vary in their preference for fertilization and sporulation sites and in the choice of host cells for sporozoites (Lainson et al. 1976, Bristovetsky and Paperna 1990). In the presently described *Schellackia*, fertilization occurs in the gut epithelium and sporogony after migration into the *lamina propria*. This differentiates the presently described *Schellackia* from *S. bolivari, S. golvani, S. occidentalis* and *S. balli* (Lainson et al. 1976, Klein et al. 1988). Another differentiating feature is the absence of sporozoites from melano-macrophages and liver hepatocytes, as is characteristic of infections with *S. landauae* (Lainson et al. 1976), *S. brygooi* (Landau 1973) *S. agamae* (Rogier 1974) and *S.* cf. *agamae* (Paperna and Ostrovska 1989). *S. ptyodactyli* sporozoites, after leaving the *lamina propria*, enter almost exclusively into circulatory white and red cells. This is reminiscent of the situation reported in *S. golvani*; although, sporozoites of the latter parasite occur only in the white blood cells.

Although experimental data are limited, whenever it has been attempted *Schellackia* spp. have demonstrated a high degree of host specificity (Lainson et al. 1976, Klein et al. 1988, Bristovetsky and Paperna 1990). *S. ptyodactyli* is the only known *Schellackia* from Geckonidae. An additional argument for creating a new species is its geographical location: the only other species of *Schellackia* known in the Middle East is *S.* cf. *agamae* from *Agama stellio*, the distiguishing characteristics of which are mentioned above.

The period of endogenous development up till the appearance of sporozoites in the peripheral blood (the prepatent period according to Klein et al. 1988) varies among the different species. Data from presently reported experimental infections of geckoes are inconsistent: a heavy infection with sporozoites in the gecko examined on day 7 p.i. evidences a very brief endogenous cycle, but it could also suggest that these sporozoites originated from an earlier, natural infection, which may have prevented the development of the superimposed parasites. Transmission in caged geckoes is, however, very unlikely to occur. In the other geckoes, gamogony as well as oogony occurred 10 days after inoculation of sporozoites, except in one gecko in which it was delayed beyond 11 days p.i. Sporozoites




**Figs. 32–35.** Transmission electron micrographs of endogenous stages of *Schellackia ptyodactyli*. **Fig. 32.** Sporozoites (arrows) in the *lamina propria*. An apical complex of one sporozoite seen extruding to the outside of the PV (c), Et = endothelial cells (× 6500). **Fig. 33.** Connective-tissue cell with three sporozoites, each situated inside a separate PV's (× 13300). **Fig. 34.** Details of a sporozoite, showing its two refractile bodies (×15,140). **Fig. 35.** Leucocyte (neutrophile?) in the *lamina propria* with several sporozoites (× 15000).

were present in the *lamina propria* of one experimental gecko on day 10 p.i. The length of the endogenous development of *S. ptyodactyli* is therefore about the same as of *S. balli* in *Bufo marinus* (Le Bail and Landau 1974), somewhat shorter than in *S.* cf. *agamae* (14–18 days – Bristovetzki and Paperna 1990), and remarkedly shorter than in *S. brygooi* (29 days – Landau 1973) and *S. landauae* (30–45 days – Lainson et al. 1976). It is also about the same as in *S. occidentalis* and *S. golvani*. Klein et al. (1988) have demonstrated, however, that lower ambient temperature (18–24°C vs. > 30°C) considerably prolonges development (from 7–17 to 30–45 days in *S. occidentalis* and from 10–12 to 21–81 days in *S. golvani*).

Hosts with chronic *Schellackia* infection seem to be refractory to experimental reinfection. Disappearance of sporozoites from the blood and liver permitted reinfection of *Agama stellio* with *S.* cf. *agamae* (Bristovetzki and Paperna 1990), suggesting preimmune-type protection. However, the immunological background for this protection phenomenon in *Schellackia* infections has not yet been investigated. In the final stages of the endogenous process, in the presently studied *Schellackia* as well as in other species (*S. brygooi, S. landauae* and *S.* cf. *agamae*), when all macrogamonts have already differentiated into oocysts and sporozoites, the gut epithelium still remains infected with variable numbers of meronts and merozoites (Landau 1973, Lainson et al. 1976, Bristovetzki and Paperna 1990). It may be deduced that immunity not only prevents entry or establishment of successive invasions of sporozoites (as in *Eimeria* spp.; Leathem and Burns 1967), but also holds back transformation of asexual stages into sexual ones. In the piscine coccidium *Goussia vanasi*, the short term acute phase, yielding gamogony stages, and sporulated oocysts is followed by a prolonged chronic infection comprised entirely of meronts and merozoites. Oocyst formation resumes only if the defense system becomes compromised (Paperna and Vilenkin 1995, and unpublished). Of all stages, sexual stages of *Eimeria* have been shown to be the most susceptible to immune inhibition (Rose and Hesketh, 1976).

Ultrastructural study confirms the already established affinities between *Schellackia* and species of *Eimeria*, and the conformity between *S. ptyodactyli* and the only other ultrastructurally studied *Schellackia* species, *S.* cf. *agamae* (Ostrovska and Paperna 1987a, b, Paperna 1992). Only merogony by exogenesis was observed. It is, however, uncertain whether endogenous development incorporates one or more generations of merogony. In *S.* cf. *agamae* merogony is by endogenesis (Ostrovska and Paperna, 1987b) but late merogonies (by day 14 p.i.) are by both end – and ectomerogony (Paperna, unpublished). Also noteworthy is the finding of mitochondria-containing intravacuolar protrusions in presumably young macrogamonts, and the elongated pseudopodium-like protrusion extending from the surface of the meronts. The number of merozoite progeny is low (<32), similar to most other *Schellackia* spp. (except *S. landauae*, see Lainson et al. 1976, Bristovetzki and Paperna 1990): this contrasts with the very high number of merozoites yielded by *Eimeria* spp. of avian and mammalian hosts (Hammond 1973).

The observed mature macrogamonts of *S. ptyodactyli* less resembled macrogamonts of *Eimeria* spp. than do those of *S.* cf. *agamae*. Our interpretation of the fine structure of macrogamonts may, however, be biased, as only a limited number of them were observed in the ultrathin sections. They contained less amylopectin granules, and a larger number of lipid vacuoles. Large electron-dense bodies, situated within very pronounced vesicles seem to correspond to the type-2 WFB, and when observed by light microscope they were faintly eosinophilic. Others, of small and medium sizes, were located in similar fashion to type-1 WFB directly in the cytoplasm. When viewed in light microscope they appeared as dense violet staining granules.

How *Schellackia* oocysts move from the mucosal epithelial layer into the *lamina propria* remains uncertain, although this phenomenon is not unusual among coccidians. Movement of macrogamonts from one cell to an adjacent one has been observed *in vitro* but could not be demonstrated *in vivo* (Speer and Hammond 1972). In *Eimeria necatrix*, the infected epithelial cells become invasive and enter the *lamina propria* by breaking down the tissue adjacent to the infected cell (Fernando et al. 1983, Pasternak and Fernando 1984). In the present observations *Schellackia* oocysts in the *lamina propria* were embedded in a functioning host cell, which suggests that the migratory process also involves the host cell. Finding oocysts within their host-cell, inside a macrophage-like cell supports an argument that macrophages (or intraepithelial lymphocytes, see Lawn and Rose 1982, Fernando et al. 1987) may be involved in the transport of the oocysts across the basal membrane.

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