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SKIN LESION PRODUCED BY THE LARVA OF CHELADONTA COSTULATA (WILLMANN. 1952) (ACARINA: TROMBICULIDAE) AND THE FEEDING MECHANISM OF THIS PARASITE

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Abstract. During the feeding of *Ch. costulata* larva an ulcer is formed in the host skin. In the base of the ulcer there is a structure termed feeding pit, which consists of a feeding tube with central canal, and hyaline mass surrounding the feeding tube. The wall of the feeding pit is formed by the tissue response of the host. We have demonstrated that the feeding tube is a product of the parasite. It is formed by the transformation of sol state of the cement secretion into the gell and consists of proteins with a higher content of arginine. The hyaline mass surrounding the feeding tube is a mixture of tissue exudate and noneffective remnants of a lytic secretion of the larva. It was found to contain acid mucosubstances. The feeding tube in the feeding pit corresponds to the stylostome of other trombiculids.

Daniel and Šlais (1957) found the larvae of *Ch. costulata* on *Microtus arvalis* Pallas 1778. The larvae induced changes in the abdominal wall of the host leading to the formation of ulcers in which the larvae were embedded. This exceptional way of parasitism is often termed "endoparasitism". In Trombiculidae it has been previously reported to occur in the genera *Hannemania* and *Endotrombicula* parasitizing the amphibians.

Most of the cases of this endoparasitism have been reported from tropical regions. In Europe it was first described by Daniel and Šlais (1957) with the species *Ch. costulata* (named *Euschöngastia ulcerofaciens*). The authors ascribed it to the intradermal mode of parasitism.

Ch. costulata larvae feed more vigorously and consequently the host tissue response is more pronounced than in most of other species of the family Trombiculidae. Daniel and Šlais (1957) described the ulcer formed during the feeding of *Ch. costulata*. In the base of this ulcer is a special structure — feeding pit. Its wall consists of a necrotic layer of connective tissue and cells of a demarcation rim. The cavity of the feeding depression is filled with a homogeneous substance. A tube with central canal is running through it. This is the only part of the feeding pit which conforms to the stylostome arising during feeding of other trombiculids.

MATERIAL AND METHODS

The material was collected during investigations of natural foci of haemorrhagic nephrosonephritis in Eastern Slovakia. The locality was characterized in detail in the paper by Daniel and Šlais (1957). The studied material, samples of skin with ulcers containing parasites, was taken from *Microtus arvalis*. It was fixed with various fixatives: 10 % neutral formalin, Susa, Zenker, 100 % alcohol, Carnoy's fluid, sublimate. After fixation the skin samples were embedded in paraffin and cut into series of 7—10 μm thick microscopic sections. For the general morphological studies the paraffin sections were stained after the following histological methods: Weigert's s Böhmer's haematoxylin, Mallory's haematoxylin, Gomori's method for impregnation of reticular fibres, Mason's trichrome and Goldner's trichrome. The detection of mucosubstances was carried out by the following methods: staining with Best's carmine in combination with saliva test and PAS reaction in combination with

acetylation, desacetylation and saliva test (Pearse 1960). Neutral and acid mucosubstances were detected by staining with alcian blue followed by PAS (AB PAS) reaction after Mowry (1963). Acid mucosubstances were detected using the staining with alcian blue (AB) at pH 2.6 (Scott et al. 1964, Quintarelli et al. 1964), with alcian blue in combination with methylation (Fisher and Lillie 1954), and demethylation (Spicer and Lillie 1959). Methylene blue extinction method (MBE-test, Pearse 1960) and critical concentration of electrolyte method (CEC) modified by Quintarelli and Dellovo (1965) were used for further differentiation.

Some amino acids and the groups containing them were detected by the following methods: Sakaguchi's method for the detection of arginine, Morel-Sisley diazotization method (Pearse 1960) for the detection of tyrosine. Tryptophan was detected by the method applying dimethylaminobenzaldehyde (DMAB) after Adams (1959). SH groups were determined by means of 2.2-dihydroxy-6,6-dinaphtyldisulphide (DDD) method of Barnet and Seligman (1952) controlled by a blockade with N-ethylmaleinimide. DDD method in combination with thioglycolic acid after Barnet and Seligman (1954) was used for the detection of SH groups. SS groups were also detected with the reaction with performic acid and alcian blue (PFA-AB) (Pearse 1960).

The copulation tetrazonium reaction modified by Müller and Chytil (1962) was used for the detection of tyrosine and tryptophan. Lipids in frozen gelatine sections were detected by Sudan black B, acid haemateine after previous treating with potassium dichromate and Fettrot 7M (Pearse 1960). Chloroform methanol extraction (Pearse 1960) was used for the control. Lipids were also detected by the reaction with α-naphtylamine complex of osmium tetroxide (OTAN) (Adams 1959, 1965) controlled by the blockade with periodic acid after Elleder and Lojda (1968) and with chloroform extraction.

Staining methods using Sudan black B and Luxol blue were used for the detection of phospholipids in the paraffin sections.

An electron microprobe was used for the stereoscopy of skin surface with ulcers and for the detection of surface distribution of calcium.

RESULTS

The larvae of *Ch. costulata* were located mainly in the posterior part of ventral region, around the genital and rectal pore.

Various stages of skin lesions caused by the *Ch. costulata* larvae were observed in the

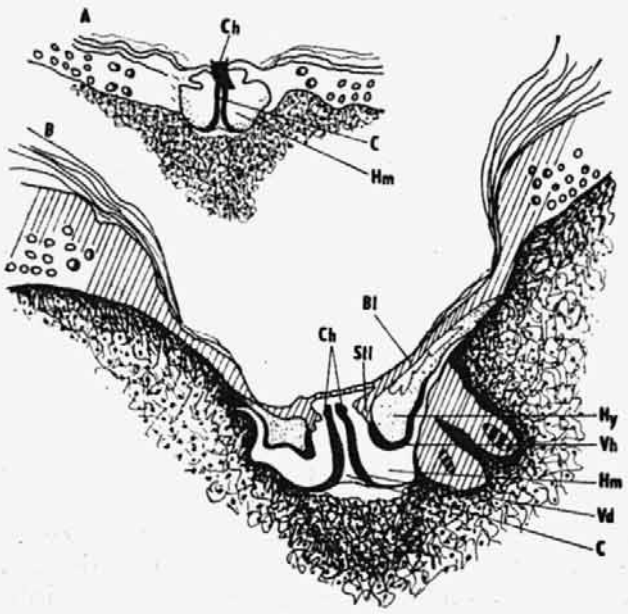

Fig. 1. Schematic diagram of gradual development of skin lesion. A — early stage of the lesion is confined to epidermis; B — upper layers of dermis are afflicted by the process at this stage.

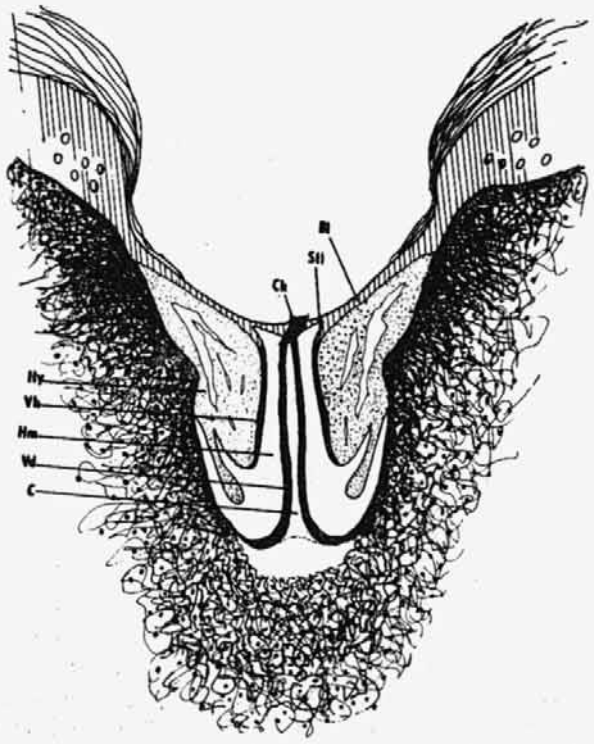

Fig. 2. Greatest development of the ulcer, pathological changes occur in the whole layer of dermis.

studied material. They could be arranged in a sequence elucidating the development of skin lesions (Figs. 1A and B, Fig. 2). In the initial stage only the epidermis of the skin is damaged (primary lesions). Later the upper layers of dermis are affected and a hypertrophic tissue rim is formed around the parasite. This lesion may be termed primary ulcer. In the following stage, the pathological changes are observed in the whole dermis; the ulcer in which the larva is embedded is at the highest stage of growth. After the release of the empty ulcer is gradually healing. The whole period of ulcer development up to its disappearance lasts about three weeks.

The character of skin may considerably influence the development of the pathological process. At the places where the layer of subcutaneous tissue is thicker, as under mammary gland, the development of the ulcer is different from that at places where the subcutis is not so markedly developed.

A. DEVELOPMENT OF LESION ON THE SKIN WITH THICK SUBCUTIS

a) Primary lesion

The initial penetration of the larva through the host skin is facilitated by chelicerae (Plate I, Fig. 1). Their movement and attachment are controlled by cheliceral muscles. The chelicerae separate stratum corneum from stratum lucidum which is then broken off by the distal ends of chelicerae giving rise to a 10 μm wide gap. The blades of chelicerae are directed towards the separated parts of stratum lucidum.

In the first phase the larva injects a lytic secretion into the gap. The cells of epidermis are disintegrated by this secretion and then sucked by the mite. The effect of the lytic secretion is so strong that the whole layer of the epidermis is always disintegrated (Plate I, Fig. 2). Immediately after the first feeding a small cavity arises in the epidermis into which flows along the chelicerae another secretion of saliva glands — cement secretion. It fills the space between stratum corneum and stratum lucidum and is further pressed between the cells of stratum germinativum. The chelicerae are cemented by this secretion at the puncture site. A tubular structure termed feeding tube (Plate I, Fig. 3) runs from chelicerae inside the skin and is gradually widening. As it is visible in the skin section showing this early stage of lesion, the cement secretion forms a lentil-like structure around the chelicerae covered by a layer of stratum corneum on its surface (Plate I, Fig. 4). The stratum lucidum is swollen up and gradually passes to the homogeneous mass of the feeding tube. The central canal of this feeding tube begins between the chelicerae, widening in form of a funnel towards the uppermost layers of dermis. The wall of the feeding tube in the direction to canal lumen is covered with an inner border layer of homogeneous character. Its structure is similar to that of the remaining portion of the tube wall, differing only in the affinity to stains. For example, it is of eosinophilic character when stained with haematoxylin and eosin, whereas the wall proper of the feeding tube is rather basophilic. The substance of the feeding tube wall has a homogeneous and hyaline appearance. Refractile fibrils, tonofibrils from disintegrated layers of epidermis, are accumulated towards the periphery of the feeding tube, at its base and on its sides.

Necrotic debris of epidermal cells are cemented in the mass of the feeding tube on its outer border (Plate I, Fig. 3). The adjacent zone of necrotic epidermis is followed by the zone of early distrophic changes and finally by the undamaged tissue of epidermis. The epidermis bordering the defect in this stage is already thickened and mitoses appear in the stratum basale.

The inflammatory reaction in the dermis is manifested by an edema and cellular infiltration formed primarily by neutrophilic and eosinophilic leukocytes. There occur also lymphoid elements and a striking activation of histiocytic cells.

b) Primary ulcer

A further stage of changes is the lesion which has already a character of ulcer (Plate I, Fig. 3). After a non-lytic secretion follows another, lytic secretion of salivary glands of the mite. The lytic secretion affects now also the cells of dermis and at the same time rises around the feeding tube to the surface layers of the epidermis. The compact connection between the mass of the feeding tube and epidermis is damaged by the lytic activity. The stratum corneum and stratum lucidum fuse with the necrotic cell debris of other layers of epidermis and form a membrane in which the chelicerae are anchored. This membrane (Plate I, Fig. 3) covers the formed feeding pit separating it from the parasite.

The epidermal cells disintegrated by the lytic secretion are sucked up by the parasite through the central canal. This action gives rise to a cavity which is filled with tissue exudate. Then again follows the phase of non-lytic secretion. The cement secretion is pressed into the space filled with tissue fluid most probably at the state of sol. The flow of the cement secretion widens in form of a funnel on the bottom of the feeding pit and spreads in a thin layer on its walls. In a histological preparation showing a longitudinal section through this primary ulcer the feeding pit with the funnel-shaped feeding tube running through its centre may be seen (Plate I, Fig. 3). A mass of hyaline appearance fills the space between the feeding tube and the wall of feeding pit. The feeding tube possesses an outer border layer separating it from the surrounding hyaline mass. This layer has similar properties as the inner border layer mentioned above. Both border layers are formed probably at the contact of the cement secretion with the liquid component of the feeding pit (Plate I, Fig. 4).

At this stage, the feeding pit is surrounded by an edematous tissue. The necrotic membrane (Plate I, Fig. 4) covering the feeding pit passes to a necrotic epidermis, which is saddle-shaped and sagging in the vicinity. The necrotic epidermis gradually passes to hyperplastic epidermis which is several times higher than normally. Also the cells of hyperplastic epidermis adjacent to the necrotic layer start to degenerate. There arise vacuoles deforming the nucleus. The wall of the feeding pit in the dermis consists of a necrotic tissue formed first by a conspicuous rim of nuclear basophilic bits followed by a demarcation border of mostly eosinophilic leucocytes and lymphocytes and further by a zone of edematous tissue in which activated forms of histiocytes and fibroblasts may be discerned. Occasionally also plasmatic cells are visible.

c) Fully developed ulcer

Simultaneously with the development of edematous and inflammatory changes in the host skin induced by the formation of the feeding pit the body of the parasite is gradually covered by a hyperplastic epidermis which is rising as a result of inflammatory edema of the dermis. In this more advanced stage of ulceration, the mite is so covered by the ulcer tissue that at most the caudal end of its body may be seen at macroscopical observation (Plate II, Fig. 1).

The histological picture of these more advanced ulcers differs in some features from the hitherto described lesions. A marked hyperkeratosis of epidermis occurs in the crater-like opening on the top of the ulcer. The hyperplastic epidermis in the margin of the ulcer is still higher than in the stage of primary ulcer. Under the hyperplastic epidermis there is a layer of hyaline degenerated collagenic fibres cemented near the necrotic wall of the feeding pit. No nuclei of fibrocytes of connective tissue cells are visible in this zone. Towards the periphery, the collagenic fibres recover their normal structure. This part of epidermis is pressed by the edema and cellular infiltration above the level of the feeding pit and it hardly reaches its first third. In this stage, the feeding pit is more elongated

and conical. The feeding tube takes the same course through the middle of the feeding pit, but sometimes there are septa visible in it (Plate II, Fig. 2) which are the remnants of the stage when the cement secretion passed from the end of the feeding tube to the walls of the feeding pit. During the repeated injection of the lytic secretion this connection was damaged and the septa remained on the bottom of the feeding pit according to the stage of coagulation of the cement secretion. The feeding tube was further elongated during the repeated injection of the cement secretion. The remnants of the septa separating the coagulated cement secretion from the feeding tube and their distance determine the succession and extent of feeding and secretion periods. In the course of the above described process the feeding pit, as well as the ulcer, become still deeper and the edema and infiltration of the whole structure are progressing. Due to a pronounced hyperplasia of the epidermis and increased cornification in the ulcer opening the larva is almost enclosed in the cavity of the ulcer. When the ulcer attains its maximum development, the feeding pit reaches up to subcutaneous muscles. The septa visible on the feeding tube increase in the size towards the bottom of the feeding pit. The conical narrowing of the feeding pit is very conspicuous in fully developed ulcers. The amount of the hyaline mass surrounding the feeding tube is decreasing. When the feeding pit penetrates through the whole dermis and reaches up to the subcutaneous muscles, the amount of the hyaline mass is minimal and it forms only a narrow basophilic ring around the end of the feeding tube. The dermis around the feeding tube is very swollen and the epidermis, due to its strong hyperplasia, nearly closes the crater of the ulcer. The demarcation rim is very wide and conspicuous and striking is the presence of cells of hystiocytic and lymphoid type.

d) Empty ulcer

At this stage the larva is released from the ulcer, but the described structures in the feeding pit persist, which indicates that they are coagulated (Plate II, Fig. 3). In the final stage, when the ulcer is healed, these structures separate together with the necrotic portion of the skin. As soon as the skin is no more irritated by the larval secretions, the edema of the tissue disappears, the swollen skin intensively proliferates and grows under its drying necrotic portion starting from the margin. The granulation tissue in the neighbourhood of the ulcer gradually ripes and has a scar character. Characteristic of this stage are the parallel collagen fibres under the epidermis covering the defect.

B. DEVELOPMENT OF THE LESION IN SKIN WITH A THIN SUBCUTANEOUS LAYER

If the larva has attached to the skin at the place with a thin subcutaneous layer, the development of the ulcer is somewhat modified.

Already after the damage of the epidermis in the first stage it is apparent that the lytic secretion of the larval salivary glands produces a rather wide than deep effect. The wall of the feeding tube is very thick and it does not grow in length very quickly. Also the necrosis of the epidermis occurs in a wide area, as well as the damage of the stratum corneum under the necrotic epidermis. The edema of the stratum corneum is not so pronounced and the dermal infiltration is limited to a narrow demarcation rim only. The feeding pit formed in the stratum corneum is shallow and widened (Plate II, Fig. 4). A hyaline degradation of collagen fibres occurs in the stratum corneum. The feeding larva remains on the surface of the skin and is not so perfectly overlapped by the hypertrophic, proliferating margin of the epidermal defect. Only in the later stage, when the skin lesion deepens the defect resembles more the ulcers arisen in the skin portion with thick layer of subcutaneous tissue.

Table 1. Results of histochemical tests for the detection of mucosubstances in the feeding pit.

Method	I central canal (lumen)	II inner border layer	III wall of feeding tube	IV outer border layer	V hyaline mass	VI tissue reaction of host
PAS	–	–	–	–	–	+++
Saliva test + PAS	–	–	–	–	–	+++
Acetylation + PAS	–	–	–	–	–	–
Desacetylation + PAS	–	–	+	–	(+)	++++
Schiff	–	–	–	–	–	–
AB + PAS		AB	PAS	AB	AB	PAS
	–	+	+	+	+++	+++
AB pH 2.6	–	+	–	++(+)	++	–
Methylation + AB pH 2.6	–	–	–	–	–	–
Demethylation + AB pH 2.6	–	–	–	–	–	–
CEC (AB pH 2.6 + $MgCl_2$)	–	10 %	–	10 %	12 %	–
Best	–	–	++	–	–	+
Saliva test + Best	–	–	–	–	–	–
MBE	–	3.6	5.6—7.8	4.7	1.5	6.8
Hale	–	dark blue	dark blue	dark blue	light blue	0
Hale + Van Gieson	–	blue	yellow-green	yellow-green	blue	yellow-green
Hale + mucicarmine	–	dark blue	dark blue	dark blue	light blue	violet
Hale + PAS	–	red-brown	red PAS	red-brown	blue	yellow-brown

Table 2. Results of histochemical tests for the detection of proteins in the feeding pit.

Method	I central canal (lumen)	II inner border layer	III wall of feeding tube	IV outer border layer	V hyaline mass	VI tissue reaction of host
Sakaguchi	–	+++++	+++++	+++++	–	–
Morel Sisley	–	++++ (+)	+++	–	(+)	+(+)
DMAB	–	++	–	–	–	+++
Copulation tetrazonium reaction	–	++++	+++(+)	+++	+	+++
DDD	–	+++	++	+++	+(+)	+++
Thioglycolic acid + DDD	–	++++	++(+)	++(+)	++	++++
N-ethylmaleimide + DDD	–	–	–	–	–	–
PFA — AB	–	–	–	–	++	–
AB pH 0.2	–	–	–	–	+++	–
Lugol + aldehyde fuchsine	–	–	–	(+)	(+)	–
Peracetic acid + aldehyde fuchsine (PAA AF)	–	+	–	+	++	+++
$KMnO_4$ + aldehyde fuchsine	–	–	–	–	++	+++

Table 3. Results of histochemical tests for the detection of lipids.

Method	I	II	III	IV	V	VI
	central canal (lumen)	inner border layer	wall of feeding tube	outer border layer	hyaline mass	tissue reaction of host
OTAN	–	red	–	red	reddish	red-black
Chloroform-methanol extraction + OTAN	–	–	–	–	–	–
NaOH + OTAN	–	–	–	–	–	red-brown
Periodic acid + OTAN	–	–	–	–	–	violet-black
Fettrot 7 B	–	–	–	–	–	red
Chloroform-methanol extraction + Fettrot 7 B	–	–	–	–	–	–
Acid hematein	–	black	–	black	–	black
Chloroform + methanol extraction + acid hematein	–	–	–	–	–	–
Sudan black B (gelatine)	–	black	–	black	–	black
Sudan black B (paraffin)	–	black	–	black	–	black
Luxol blue (paraffin)	–	blue	–	blue	–	blue

C. HISTOCHEMISTRY OF SKIN LESION PRODUCED BY *CH. COSTULATA* LARVA

The results of histochemical studies elucidating the histochemical nature of the feeding pit are summarized in Tables I—III.

Individual parts of the feeding pit exhibit a different affinity to stains which indicates the heterogeneity in its chemical structure. The following layers may be distinguished: the inner border layer, the wall proper and outer border layer of the feeding tube, hyaline mass surrounding it and wall of feeding tube formed as a tissue response of the host.

Acid mucosubstances with sulphogroups (SO_3H), proteins containing sulphydryl groups (SH), amino acids tyrosine, tryptophan, histidine and a large amount of arginine were found in the inner border layer. The wall of the feeding tube contains a high percentage of protein components — arginine, tyrosine and histidine. The outer border layer contains acid mucosubstances and it is rich in arginine. The hyaline mass surrounding the feeding tube contains particularly acid mucosubstances with sulphogroups (SO_3H) and a small amount of SH and SS groups. The zone of tissue reaction contains neutral

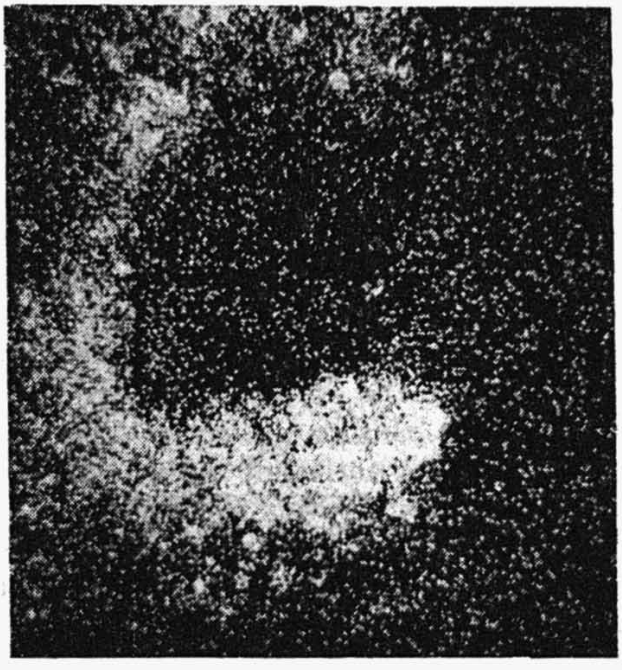

Fig. 3. Linear analysis of calcium distribution in transverse section through the bottom of ulcer. Maximum calcium distribution in the zone of tissue reaction. (Current intensity $3 \cdot 10^{-9}$, accelerating voltage 20 kV: 150×).

mucosubstances and of the protein components, tyrosine, histidine and a higher amount of tryptophan, and a high concentration of SH and SS groups. The presence of calcium was detected in this zone by a physical method of microanalysis using electron microanalyzer JXA-5 (Fig. 3).

DISCUSSION

The skin lesion provoked by the larva of *Ch. costulata* differs from the stylostome arising during the feeding process of other larvae of the family Trombiculidae. The lesions provoked by the latter are mostly confined to epidermis and partly corium and usually do not spread to hypodermis (Šlais 1960). However, the structure arising during the feeding process of the *Ch. costulata* larva and termed feeding pit reaches deep into the hypodermis. It is formed by alternating injections of lytic and cement secretions of salivary glands of the larva. The histochemical methods revealed that the wall of the feeding tube running through the middle of the feeding pit is of rather proteinaceous character and contains arginine, tyrosine, tryptophan and histidine. The arginine is evenly distributed in the whole wall of the feeding tube, whereas tyrosine, tryptophan and histidine are much more concentrated in the inner and outer border layers. Except arginine, these amino acids were detected also in the region of tissue reaction so that it is possible that the amino acids detectable, e.g., in the inner border layer of the feeding tube originate just from this zone.

The feeding tube is surrounded by a hyaline mass which is rich in mucosubstances. These are, however, also in the outer and inner border layers of the feeding tube. We assume therefore that the mucosubstances may get here from the hyaline mass from the vicinity of the feeding tube like some amino acids penetrating here from the zone of the host tissue reaction.

While comparing histochemical reactions of the feeding tube of *Ch. costulata* with those of the stylostome of other Trombiculidae (Schumacher and Hoeppli 1963) it may be said that the comparable anatomical structures (stylostome and feeding tube; region of tissue reaction to *Ch. costulata* secretion) exhibit identical reactions. There are some differences between the results obtained by Voigt (1970) with trombiculids and our results with *Ch. costulata* as regards the staining ability. At the places where Voigt observed an uneven staining of layers, we have often seen an even staining of the whole layer in ulcers provoked by *Ch. costulata*.

Of interest is the formal genesis of the feeding tube. The initial cavity of the feeding pit arises by disintegration of epidermal cells, and later also of dermis, by the lytic secretion. Another secretion is then injected into the cavity and fills it up. At the same time, already at the beginning of the process, the central canal is formed. Its origin is explained by the fact that the cement secretion at the state of sol is pressed into the cavity and is gradually transformed to gel. Already Schumacher and Hoeppli (1963) supposed the transfer of the cement secretion from the sol to the gel phase. This transfer is initiated by the contact of the secretion with the tissue or tissue fluid of the host. The reaction gradually occurs in the whole mass of the secretion starting from its periphery, where it is in contact with the host tissue, towards its centre. Since in the centre the sol is permanently moving, being connected with the mouth opening of the larva, this change cannot occur and the central canal is thus arising.

After the injection of the cement secretion follows again the injection of the lytic secretion disintegrating the dermis and even the continuous connection of the hyaline mass forming the feeding tube with the epidermis. In this it differs from the stylostome of other trombiculids, where the continuous transfer between the stylostome substance and epidermis persists even after a longer lytic secretion.

During the sucking of the disintegrated tissue the tissue fluid is flowing from the surrounding tissue into the cavity. As soon as the feeding is finished, the space of the cavity is filled with this fluid and the cement secretion at the sol state is again forced through it. Due to this "jet flow" effect the secretion penetrates up to the distal end of the feeding pit. During this process it gets into contact with the fluid which again initiates its transfer to gel. The alternating flow is again pressed up to the bottom of the pit where it closely adheres to its walls and forms a funnel-shaped widening.

Our explanation of the origin of the feeding pit by alternating injections of two types of secretion conform to the idea of Schumacher and Hoeppli (1963) about alternation of two different secretions of trombiculid larvae. Also Voigt (1970) explains the origin of the stylostome and its elongation by two alternating secretions. She assumes, however, that the lytic secretion is secernated already at the time when the non-lytic secretion is still at liquid state. In our opinion, the lytic secretion is secernated only when the cement secretion has already changed from the sol to the gel phase.

The formation of the hyaline mass surrounding the feeding tube may be explained by the accumulation and coagulation of the tissue fluid on one hand and by the accumulation of an excessive, no more effective lytic secretion on the other hand. This explanation is supported also by the positivity of this substance at the detection of acid mucosubstances.

While studying the feeding mechanism of the larva we have observed sucking of the disintegrated host tissue through the feeding tube. Remnants of disintegrated epidermis and dermis were often found in the central canal of the feeding tube. However, the larva sucks also the cells of the exudate which have immigrated to the cavity of the feeding pit from the surrounding zone of inflammatory reaction. André (1927) termd$_o$ the process during which the host tissue cells are disintegrated by the lytic secretie "extraoral" or "extraintestinal digestion". We assume, however, that it is more suitable to speak about a sort of preliminary digestion, since corpuscular cell debris are found in the central canal. For the extraintestinal digestion a complete liquefaction of the tissue would be necessary, but this occurs only in the intestine and not in the feeding tube of the larva. This conclusion is supported also by the observations of Voigt (1971) who studied the feeding of various Trombiculidae species on various hosts.

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ПОРАЖЕНИЕ КОЖИ, ВЫЗЫВАЕМОЕ ЛИЧИНКОЙ *CHELADONTA COSTULATA* (WILLMANN, 1952) (ACARINA: TROMBICULIDAE) И МЕХАНИЗМ ПИТАНИЯ ПАРАЗИТА

Я. Шрамлова

Резюме. Во время питания личинок *Ch. costulata* возникает язва в коже хозяина. В базе язвы находится строение, называемое питательная ямка, состоящая из питательной трубки с центральным каналом и гиалиновой массы, окружающей питательную трубку. Стена питательной ямки образуется тканевым ответом хозяина. Было показано, что питательная трубка является продуктом паразита и возникает переменой состояния sol цементного секрета в состояние gel и состоит из белков с высшим содержанием аргинина. Гиалиновая масса, окружающая питательную трубку, является смесью тканевого экссудата и не действующих остатков литического секрета личинки. Было обнаружено, что она содержит кислые мукосубстанции. Питательная трубка в питательной ямке соответствует стилостоме других видов Trombiculidae.

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EXPLANATIONS TO FIGURES AND PLATES

Ch — chelicerae, C — central canal, Hy — hyaline mass surrounding the feeding tube, Hm — feeding tube, Stl — swollen stratum lucidum, Bl — membrane separating the pit from the parastite, Vh — outer border layer, Vd — inner border layer

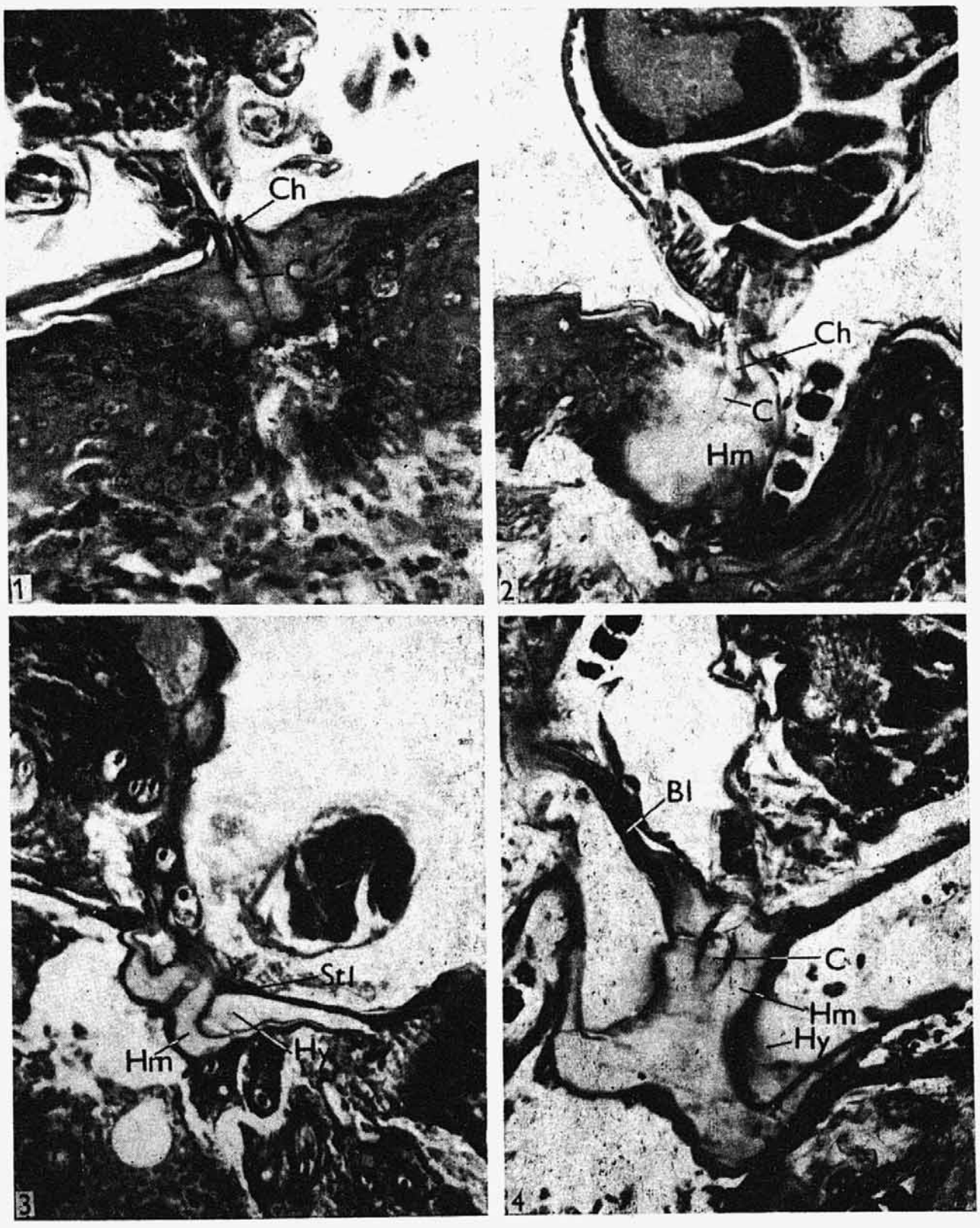

Fig. 1. Primary lesion in hyperplastic epidermis. (Hematoxylin-eosin, 400×). **Fig. 2.** Primary lesion. Note cementation of necrotic cellular debris of epidermis in faintly stained feeding tube. (Hematoxylin-eosin, 400×). **Fig. 3.** A rim starts to form around the larva and the lesion has a character of primary ulcer. The central canal is filled with eosinophilic mass. The space under the bottom of feeding pit is artificially widened by retraction during fixation of its content. (Hematoxylin-eosin, (200×). **Fig. 4.** Developed primary ulcer. Note central canal, feeding tube, hyaline mass surrounding the feeding tube and membrane covering the feeding pit. (Hematoxylin-eosin, 400×.)

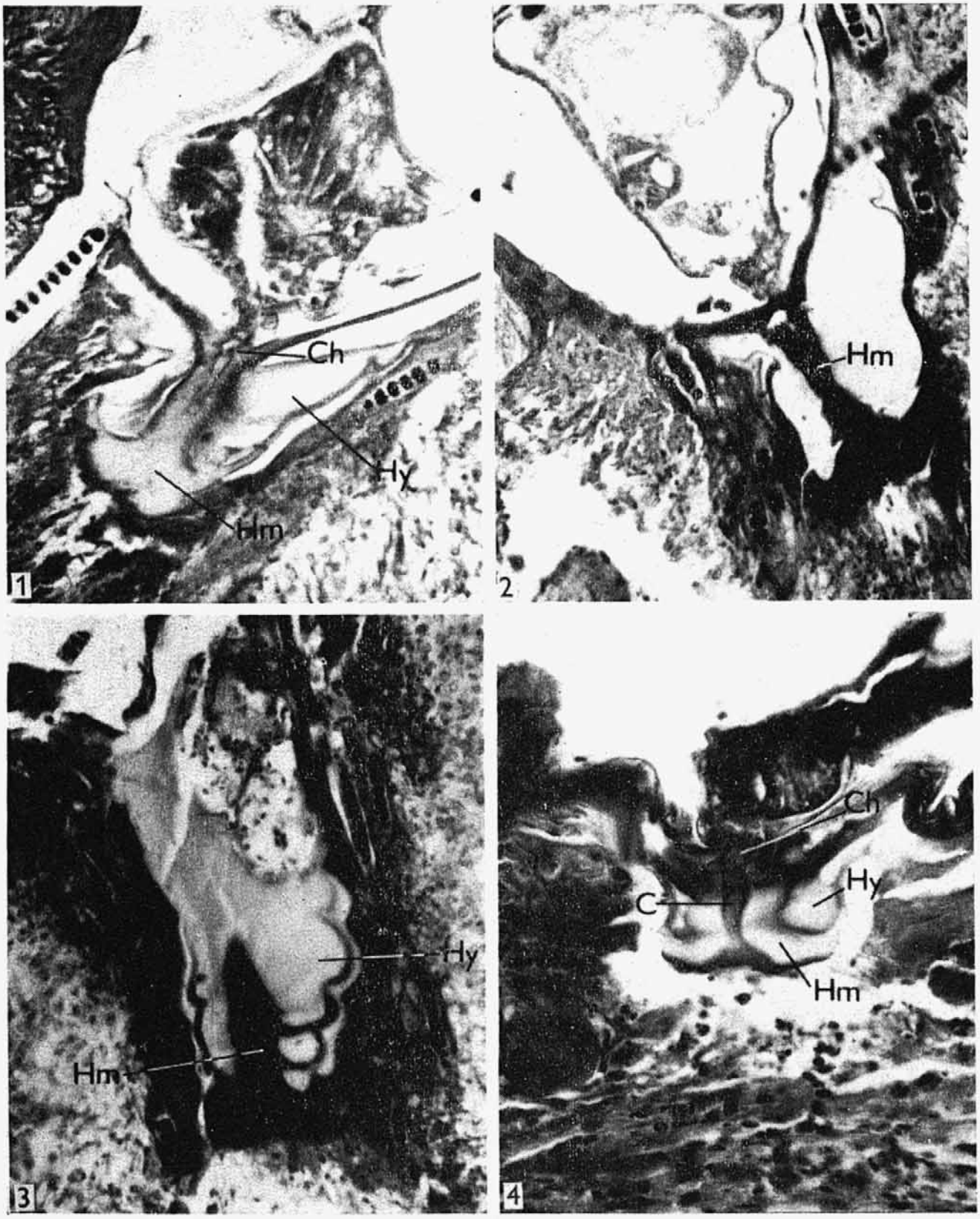

Fig. 1. Further stage of deepeding of feeding pit into dermis. (Hematoxylin-eosin, 200×). **Fig. 2.** Fully developed ulcer. (Goldner, 200×). **Fig. 3.** Empty ulcer. Note septa on the feeding tube. (Goldner, 200×). **Fig. 4.** Lesion on skin with thin subcutaneous layer. Primary ulcer. (Hematoxylin-eosin, 200×.)