stimulating secretion in mammals[4,5,11,12] and that histamine is released from isolated gastric mucosa of the frog by both acetylcholine and pentagastrin[13] strengthens this theory. Our experiments which demonstrate that metiamide is a powerful inhibitor of the acid gastric secretion which accompanies chloralose-urethane anaesthesia appears to offer further support for this view, as is our observation that 'spontaneous' gastric secretion in conscious dogs is blocked by metiamide. Despite the above facts, there are observations which indicate that the question of whether histamine is the final common mediator must be left open as yet. For example, there are reports that the acid secretion evoked by cholinergic agents in the rat[3] or dog[3,14] appears to be more resistant to blockade by H_2-receptor antagonists than is the secretion produced by histamine or pentagastrin. The possibility, therefore, that several mediators act on distinct but overlapping receptor sites in the gastric mucosa cannot as yet be excluded. Perhaps there are other quite different explanations of these facts. What is clear, however, is that H_2-receptor antagonists, perhaps in combination with anti-cholinergic agents, offer a new and effective means for the suppression of acid gastric secretion of physiological or pathological origin.

Summary. The histamine H_2-receptor antagonist, metiamide, inhibits the acid gastric secretion produced by chloralose-urethane anaesthesia in dogs carrying gastric cannulae chronically. This secretion is also prevented by atropine and hexamethonium. 'Spontaneous' gastric secretion of vagal origin in conscious dogs is also blocked by metiamide.

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[16] We acknowledge the able technical assistance of Mr. Tim Barry and Mr. Dan Fackre.

A New Glandular Organ in some Toxic Caterpillars

Previously undescribed exocrine glands associated with the tracheal system and producing a micellar lipoidal secretion are present in caterpillars of some families of Lepidoptera. The glands comprise a small number of large specialized cells lying midventrally in the third to sixth abdominal segments, slightly below and behind the ganglia of the ventral nerve cord. They are associated with and apparently derived from the nodes of the ventral transverse trachea. Tracheal nodes which are located segmentally on longitudinal trunks or medially on transverse commissures mark the anastomosis of adjacent tracheal rudiments. They have been recognized in larvae of both Diptera[1-3] and Lepidoptera[4] not only as points of anastomosis, but also as points of dehiscence that facilitate breakage and withdrawal of the tracheal lining at each ecdysis. In the larvae of most Lepidoptera the nodes occur as distinct dilations in which the tracheal intima is modified into a thin cuticle bearing inwardly directed spines. Usually the epithelium of the nodes is composed of cells that, although considerably larger than those of the adjacent unmodified tracheal epithelium, are not glandular. In larvae of some species however, the epithelium on nodes of 4 of the ventral transverse trachea is clearly modified into glandular organs. We have found such organs in caterpillars of 7 of the 20 families surveyed (Table). They are best developed in arctiids in which they occur as spheroidal clusters of very large cells.

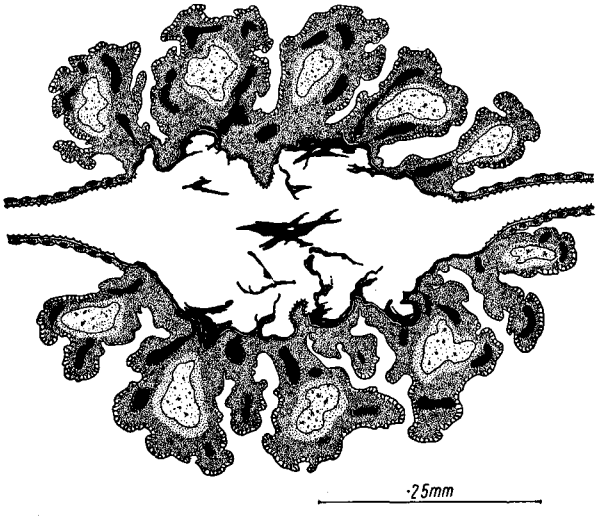

Fig. 1. Mid-ventral glandular tracheal node in 5th abdominal segment of *Pyrrharctia isabella* as it appears in section.

Histological and fine structural investigations of these glands in the banded woollybear caterpillar, *Pyrrharctia isabella* show the cells to be both highly specialized and unusual in structure (Figure 1). The prominent nuclei are large, irregularly shaped, and with sparse, evenly distributed chromatin. However, the most conspicuous feature of the cells are large, densely staining, cylindroid accumulations of secretory product. Ultrastructurally the secretion has a micellar structure with a lamellar periodicity of about 55 Å (Figure 3). It lies within an interconnected branching lacunar system that is lined with microvilli (Figure 2). The lacunar system opens at the apical end of the cell onto the tracheal lining of the gland. Although we have not found unequivocal evidence of channels or pores traversing the lining, the presence of material histochemically similar within the tracheal lumen indicates that it passes through the cuticular lining. The cytoplasm of the cells contains numerous mitochondria with dense

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Occurrence of glandular tracheal nodes in the Lepidoptera

Family	Species examined	Glands present	Family	Species examined	Glands present
Agaristidae	1	+	Noctuidae[a]	15	+[a]
Amatidae	1	+	Notodontidae	2	—
Arctiidae	6	+	Nymphalidae	3	—
Bombycidae	1	—	Papilionidae	2	+
Citheroniidae	1	+	Pieridae	2	—
Danaidae	1	—	Pyralidae	2	—
Eucleidae	1	—	Saturniidae	3	—
Geometridae	4	—	Sphingidae	4	—
Lasiocampidae	1	—	Zygaenidae	1	—
Liparidae	2	+	Hesperiidae	1	—

[a] Present in the Heliothidinae, Pantheinae and Acronyctinae, absent in other sub-families.

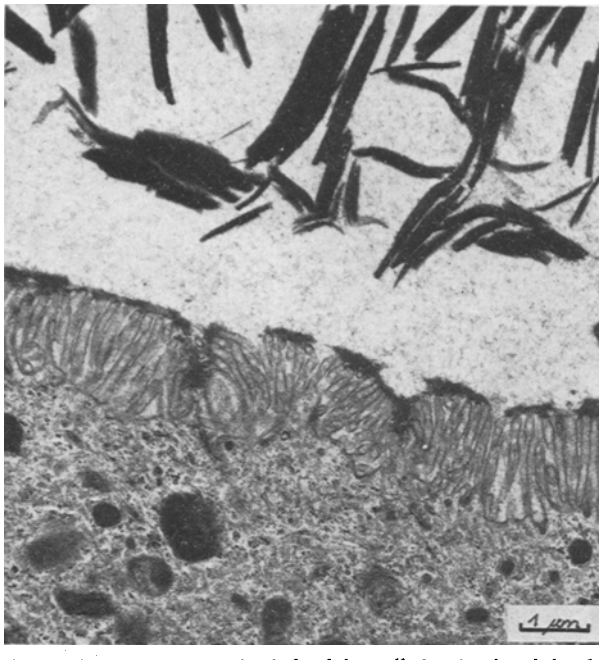

Fig. 2. Electron micrograph of glandular cell showing brush border and secretions within the lacuna. ×10,000.

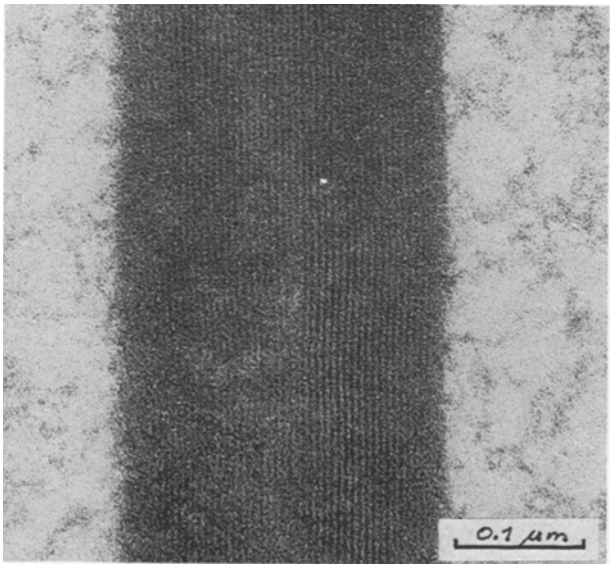

Fig. 3. Electron micrograph of secretion showing lamellar substructure. ×170,000.

matrix and relatively few cristae, a moderate number of small golgi bodies and very extensive smooth endoplasmic reticulum. The basal margin in contact with the haemolymph is characterized by extensive infoldings of the cell membrane beneath a basal lamina.

The lipoidal nature of the secretion is suggested by its lamellar substructure which is similar to the bimolecular lamellar configuration assumed by polar lipids, and this is confirmed histochemically by its osmio- and sudanophilia. However, histochemical tests[5] for phospholipids were negative as were tests for both proteins and carbohydrates. The secretion stained with paraldehyde fuchsin after oxidation, which in view of the negative tests for protein and carbohydrates would indicate that the lipoidal material is unsaturated. The secretion is soluble in acidic aqueous solutions and organic solvents but not in weakly alkaline or neutral aqueous solution.

On the basis of their ultrastructure, especially the extensively infolded basal membrane, it seems likely that these organs are involved in the removal of substances from the haemolymph. The substances are apparently conjugated to or incorporated into a lipoid synthesized or elaborated within the cells and the material thus formed transferred by a process of exocytosis to the lacunar system. As the secretion accumulates some of it passes into the trachea, presumably to be removed at ecdysis. This process, therefore, has the characteristics of storage-excretion and is unique in that it utilizes the tracheal system.

Apart from the several noctuoid families there is no close phylogenetic relationship among the Lepidoptera having these organs. Although they occur in some papilionids and heliothidines which are not hairy, they occur predominantly in larvae with an investiture of urticating hairs[6]. It is also noteworthy that they occur in groups that are generally considered to be distasteful or to have a predilection for poisonous plants, and some of the Arctiidae, Amatidae and Papilionidae at least are known to have the ability to sequester and store toxic substances[7–10].

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Although we have no direct evidence regarding the function of these organs we suggest that they are involved in the sequestration and storage of some substances from the haemolymph, and that these substances may be toxic.

Summary. The nodes formed by the mid-ventral tracheal anastomoses in abdominal segments 3–6 are modified into conspicuous glandular organs in larvae of certain Lepidoptera. They comprise clusters of extremely large cells penetrated by an extensive lacunar system opening onto the tracheal wall. These cells appear to sequester substances from the haemolymph which may be conjugated with a lipoid synthesized within them and the product excreted into the lacunar system, ultimately passing into the tracheal lumen.

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[11] Acknowledgments. We thank J. A. DOWNES and E. G. MUNROE for helpful discussions and advice.

Glycogen in Epidermal Nerve Terminals of *Lacerta sicula* (Squamata: Reptilia)

Epidermal nerve endings in reptiles were first described in turtle skin by HULANICKA[1]. She describes them to be 'conspicuous buttons, lying just beneath the stratum corneum, ovally shaped, their longer axis parallel to the skins surface'. JABUREK's[2] light-microscopical study revealed them to be located 'intracellularly'. Recent electron-microscopic work[3–5] shows that axons ascend through the intercellular space of the epidermis, penetrate the uppermost keratocytes (just beneath the α-keratin layer) and enlarge to several μm-sized 'discoid terminals' (a term which we prefer to VON DÜRING's 'bubble receptor' since they are not gas-filled). One axon can give rise to two or even more[2] discoid terminals (Figure 1). These terminals seem, in the section, to be surrounded by two membranes: the outer is part of the plasmalemm of the keratocyte, the inner, thicker one, part of the axolemm (Figure 2). They contain numerous mitochondria, and are densely filled with particles[3,5], which the UAc-PbCi staining (Uranyl acetate – Lead citrate) (Figures 1 and 2) shows to be 250–370 Å in diameter (mean diameter 300 Å). They are not membrane-bound. In comparison, free ribosomes are approx. 200 Å in size. Higher resolution (Figure 2) reveals no further details. Earlier work[3–5] presumes those particles to be glycogen. In order to verify the glycogen nature of these particles periodic acid-lead citrate (PA-PbCi) stain and α-amylase digestion were applied.

The PA-PbCi stain[6] (Figure 3) is specific for polysaccharides: Following oxidation by PA, aldehyde groups appear in the polysaccharide chains which may be detected by Pb ions. The particles are 250–350 Å in size (mean diameter 300 Å), thus corresponding to glycogen α-particles or rosettes. The high magnification (Figure 3) shows β-particles (mean diameter 70 Å, range 50–100 Å). Between the β-particles we find contrasted material (Figure 3), according to DE BRUIJN[7] nonparticular glycogen. DE BRUIJN claims it to be a separate entity composed of oligosaccharides or glycogen-enzyme complexes of the glycogen metabolism and relates it to the γ-particles described by DROCHMANS[8] (discussion see below).

Enzymatic digestion with α-amylase according to MONNERON and BERNHARD[9] brings the final proof that these particles are glycogen. Figure 4 shows the succesive stages of digestion: undigested α-particles are still recognizable; soon a brightening is observed in the center of the particle; with the proceeding expansion of the bright center the α-particle looks like a membrane-bound vesicle (these are abundantly seen); finally no trace of α-particles can be observed: large areas where particles would most likely be found exhibit only a homogenous matrix. We believe all those different structures to be stages in the digestion of the α-particle, because of the identical size of α-particles and vesicle-like structures (250–350 Å, mean diameter 300 Å) and absence of vesicle-like structures in undigested material (Figures 2 and 3). The big holes of variable diameter can be interpreted as follows: Figures 2 and 3 show that α-particles often aggregate closely contacting each other. Since the thickness of the sections corresponds to about 3 average α-particle diameters (800–1000 Å), the holes may be sites of such aggregates arranged vertically which are completely digested. Backing this explanation is the fact that the holes are often surrounded by an electron-dense boundary corresponding to the membrane-like structure of the α-particle digestion stage described above.

These electron-dense boundaries, respectively membrane-like structures, may represent limit-dextrins aggregated peripherically by what cause so ever. α-Amylase breaks α-1→4 glycosidic linkages – with preference internal ones – but cannot split the α-1→6 bonds (branching points) contained in the limit-dextrins[10]. Due to the aldehyde groups appearing following oxidation by PA, the limit-dextrin stains with Pb ions. But increased incubation brings them to disappear as seen in Figure 4, since the α 1→6 bonds may split without enzymatic activity, thus giving to the α-amylase the opportunity to break further α-1→4 linkages (glycogenolysis occurs even in control sections which are treated with trypsin – inhibitor but in a much slower manner: they need about 10 times the incubation time to show the same effect as do the sections digested by α-amylase). Our interpretation considering the electron-dense boundaries is backed by THORNELL's[11] statement that α-amylase treated sections remain stainable with PA-TSC-SP (periodic acid-Thiosemi-carbazide-Silver proteinate), which reveals polysaccharides[12]. Supposing these boundaries were glycogen-enzyme complexes which may be present between the β-particles according to DE BRUIJN[7], they would have to be visible even in extended incubation

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