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# SEQUENTIAL DEGENERATION OF THE LARVAL MUSCLES IN THE LEMON-BUTTERFLY *PAPILIO DEMOLEUS* L AND ITS POSSIBLE SIGNIFICANCE

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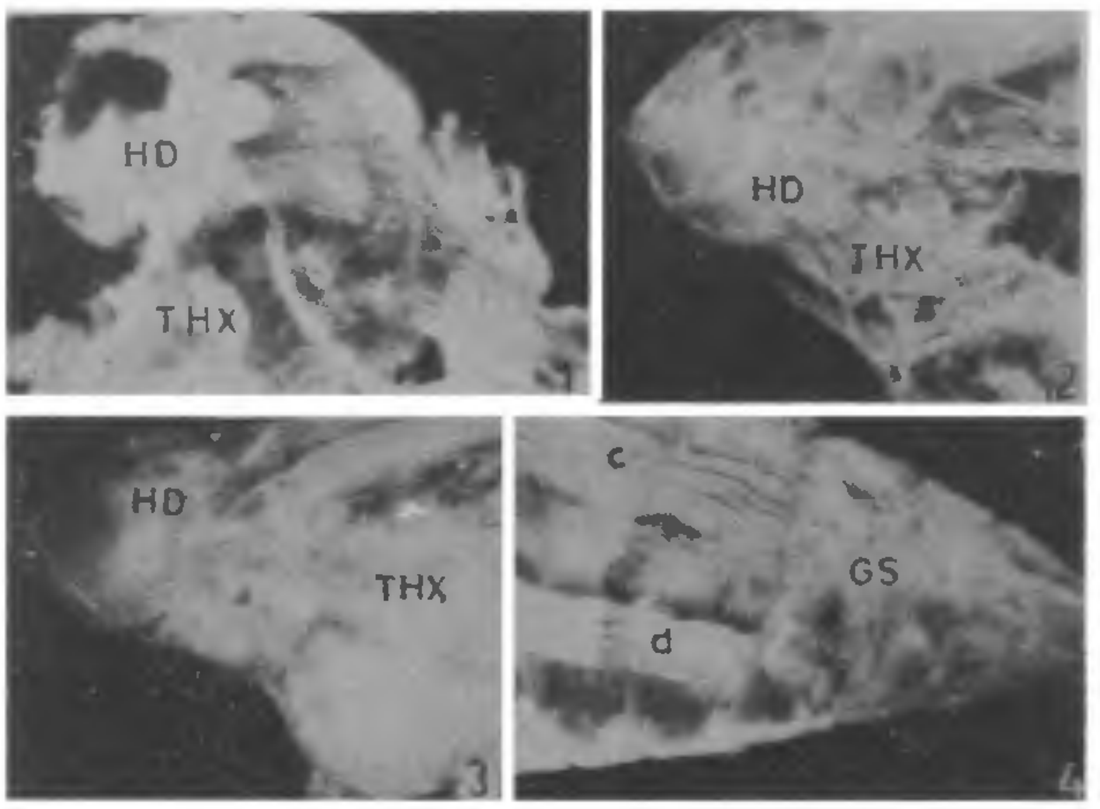
SEQUENTIAL degeneration of the larval muscles during metamorphosis was discovered by Hufnagel[1] and later rediscovered by several other workers[2–5]. However, its significance has not so far been explained. In this note we have attempted to do this.

The ultimate (fifth) instar larvae of the lemon-butterfly were sorted out from the laboratory colonies and their muscle loss was followed during the larva-pupal-adult transformation up to 1-day-old adult. The muscle degeneration was recognized by the loss of its fibrillar appearance and acquisition of a flaky one.

Degeneration of the larval muscles does not occur simultaneously in all the body segments but follows a definite sequence. For convenience, we have grouped the muscles as segmental and spiracular. Amongst the segmental muscles, the first to be lost are the cranial muscles between the prepupal (figure 1) and zero hr (newly ecdysed) pupal (figure 2) stages, next are the thoracic muscles (figure 3) and muscles of the posterior abdominal or future genital segments (figure 4) between 0 and 36 hr pupal stage followed by the muscles of the anterior abdominal or pregenital segments between 0 and 48 hr pupal stage except muscles a-e (figure 5, muscles c and d, also seen in figure 4) which survive metamorphosis and are lost in 1 day old adult. Amongst the spiracular muscles, the ventral spiracular dilator, f (figure 6) is lost in the pupal stage, the dorsal spiracular dilator, g in 1-day-old adult and the spiracular occlusor, h is not lost at all but persists throughout the adult life.

A selective or sequential muscle degeneration is probably an universal phenomenon throughout the pterygote insects[6]. However, earlier workers do not seem to have explained or speculated upon the possible significance of this phenomenon. Although, direct experimental evidence to explain it will need techniques to block or allow muscle degeneration at will and since such techniques are not immediately available, we have




**Figures 1–4.** **1.** Sagittal section of the early prepupal head and thorax prior to commencement of muscle degeneration. Note the fibrous (undegenerated) nature of the muscles. **2.** Same in zero hr pupa showing degeneration of the cranial muscles as indicated by their flaky appearance. Thoracic muscles are still fibrous. **3.** Same in 36 hr pupa showing degeneration of the thoracic muscles. **4.** Sagittal section of the abdomen of 36 hr pupa showing degeneration of the muscles of the genital (VIII–X) segments and the intact condition of those of the pregenital (I–VII) segments.

depended on circumstantial evidence to explain this phenomenon as follows: The sequential muscle degeneration seems to be associated with the complexity of the (adult) structures that replace the larval ones postmetamorphosis. For instance, the biting and chewing mouthpart-bearing head of a caterpillar, whose movements are restricted to its own plant host as in the present case, will need drastic remodelling to convert itself into a nectar feeding head of an adult butterfly that not only needs sucking mouthparts but also a pair of efficient compound eyes to meet the requirements of its actively flying habit. Likewise, the thoracic and genital segments whose muscles degenerate next to those of the cranial segments have to undergo a much greater degree of remodelling to acquire structures like wings and external genitalia respectively than the pregenital segments which remain unmodified except for their hardening and scaly covering and therefore are last to lose their muscles. If we have to seek a reason/significance for this order of change, one has to speculate that greater the degree of remodelling, longer the time required to achieve it and so earlier its commencement to beat the deadline of metamorphosis. For the significance of the survival of muscles a–e (figure 5) up to 1-day adult, Finlayson[3, 6] explained that these muscles are needed to pump blood into the wings to help them expand and therefore, they must stay until adult emergence and some time thereafter. However, the loss of spiracular muscles is not

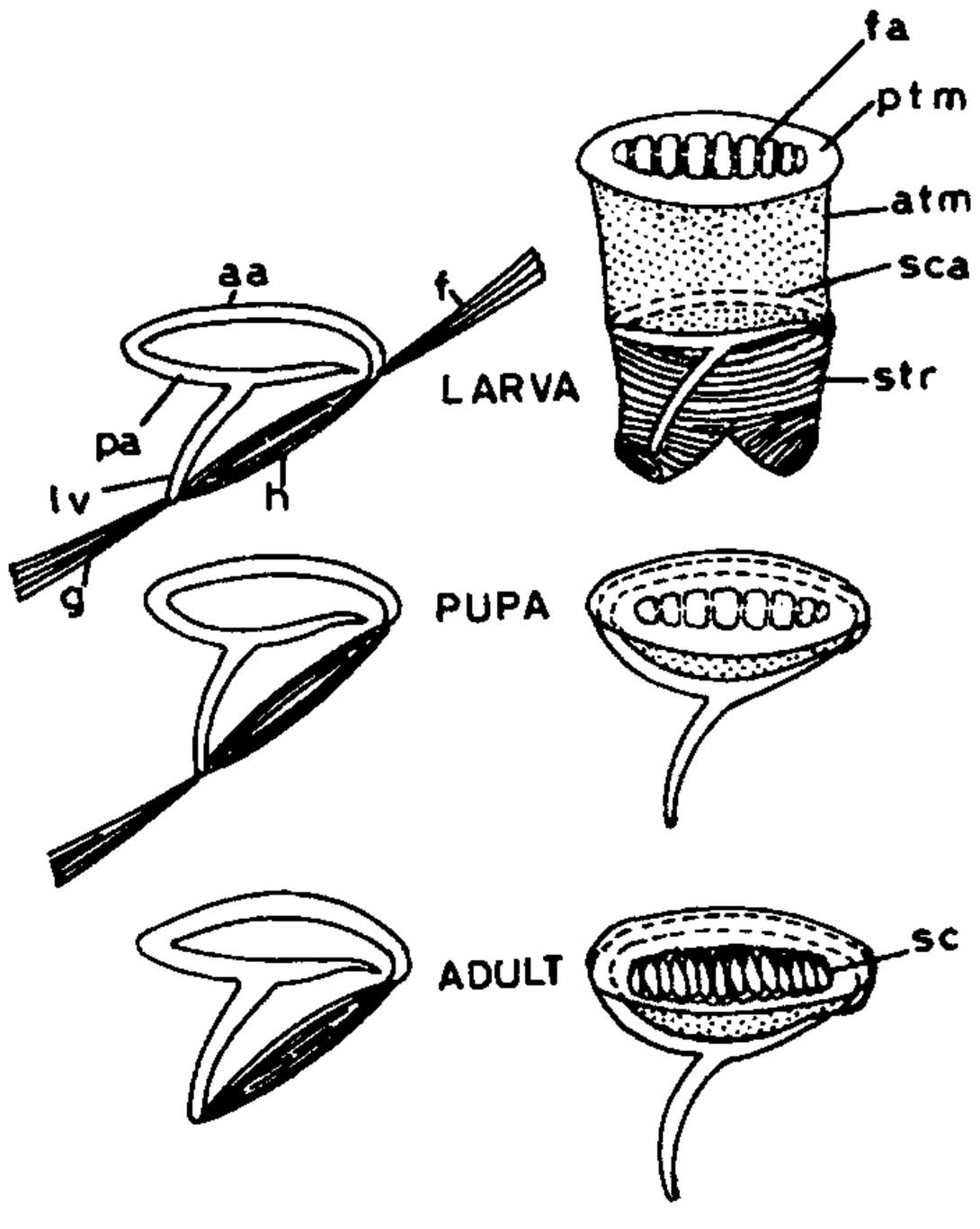



**Figure 6.** Metamorphic changes in the spiracular mechanism. Figures on the right show obliteration of atrium and consequent fusion of the anterior-half of the spiracular closing apparatus with the peritreme while those on the left show the accompanying loss of the spiracular muscles.

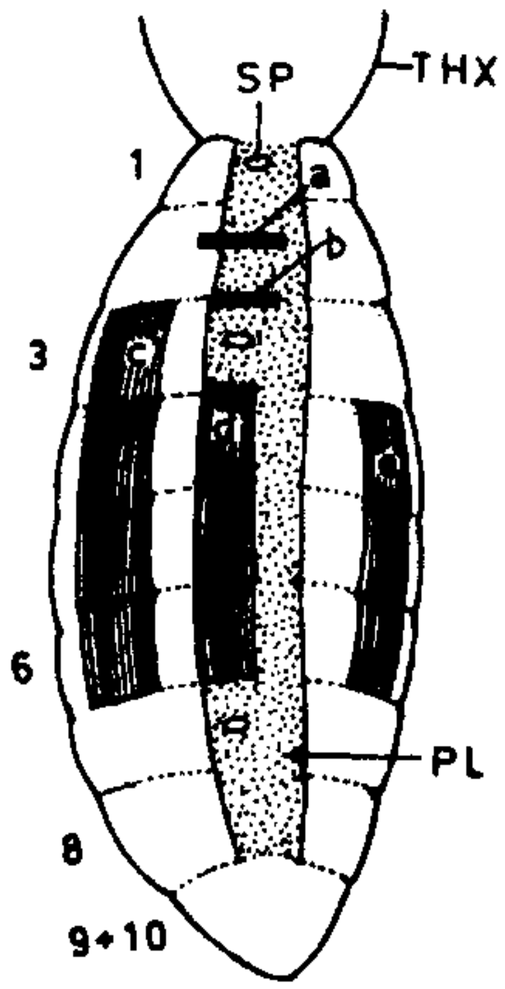



**Figure 5.** Sagittal section of the abdomen of a newly emerged adult showing muscles (a-e) that survive metamorphosis to be lost in 1-day-old adult.

**Abbreviations used in figures 1–6.** a-e: muscles surviving metamorphosis; aa: anterior-half of the spiracular closing apparatus; atm: atrium; f-h: spiracular muscles; fa: filter apparatus; GS: genital segments; HD: head; lv: lever; pa: posterior-half of the spiracular apparatus; PL: pleuron; ptm: peritreme; sc: scales replacing filter apparatus; sca: spiracular closing apparatus; SP: spiracle; str: spiracular trachea; THX: thorax; 1-10: abdominal segments.

complexity-based but seems to be related to the position of the spiracular closing apparatus (see Srivastava[7]) in the larval and adult stages. In the larva, this apparatus stays free in the body cavity away from the body wall due to the intervention of a prominent membranous atrial region. Such a suspended closing apparatus will obviously require two antagonist forces in the form of dorsal and ventral spiracular dilators to open up the spiracles. A single dilator, as can be seen,

will pull the apparatus on its own side without effecting spiracular opening. This situation changes in the pupal stage. The anterior-half of the closing apparatus comes to fuse with the peritreme, a sclerite in the body wall surrounding the spiracular opening (orifice) due to the obliteration of the atrium and is thus rendered immobile. The ventral spiracular dilator which has its insertion on this part of the closing apparatus is thus rendered redundant and is therefore lost in the pupal stage. The question of the loss of the dorsal spiracular dilator in 1-day old adult can be answered only if we assume that the free posterior-half of the closing apparatus by this time acquires a degree of (intrinsic) elasticity that is adequate to open the spiracle by its own force. The assumption gains credence since it tends to explain the permanence of the spiracular occlusor. For, if this muscle were also to be lost, the intrinsic elasticity of the free half of the spiracular closing apparatus would keep the spiracles permanently open and thus destroy the spiracular regulatory mechanism.

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## SEASONAL MORTALITY OF *DIADEGMA TRICHOPTILUS* (CAMERON) (HYMENOPTERA: ICHNEUMONIDAE), A LARVAL PARASITOID OF *EXELASTIS ATOMOSA* WALS DUE TO HYPERPARASITOIDS

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IN biological control, care must be taken to prevent the introduction of hyperparasitoids in fields and is based on the notion that hyperparasitoids may seriously affect the efficacy of primary parasitoids[1]. Keeping this in view the present work was carried out.

During a survey for hyperparasitoids in 1980–1983 in Marathwada, extensive cocoon collection of *Diadegma trichoptilus* (Cameron) (Hymenoptera: Ichneumonidae), a larval parasitoid of *Exelastis atomosa* Wals pest of pigeon pea (*Cajanus cajan*. Mill) was made. At laboratory condition ($24 \pm 11$ °C, 55% R.H.) the cocoons were kept in 5 per test tube for adult emergence. The emerged parasitoids and hyperparasitoids were recorded.

The mortality of 2421 *D. trichoptilus* cocoons was due to three hyperparasitoids: (i) *Brachymeria* sp (Chalcidae); (ii) *Eurytoma* sp *braconidis* group (Eurytomidae); (iii) *Paraphylex* complex (Ichneumonidae).

The seasonal mortality averaged 4.94% in November, 33.20% in December and 62.90% in January. The hyperparasitoids appeared 1–2 weeks later than the parasitoid. The percentage of hyperparasitization increased with increase of parasitization by *D. trichoptilus*. In *Trioxys* (*Binodoxys*) *indicus* Subba Rao and Sharma, hyperparasitization declined due to fall in temperature and humidity and increased with rise of temperature[2]. In *Apanteles congregatus* (Say) hyperparasitization by *Hypopteromalus tabacum* (Fitch) increased during cold weather[3]. Similarly in the present study hyperparasitization increases with lowering of temperature. Hyperparasitization had a peak value of 62.9% in January but as the harvesting period of host crop approached, the emergence of hyperparasitoids stopped. This indicates that hyperparasitoids have entered in diapause. With *Cotesia orientalis* Chalikwar and Nikam similar results were noted[4].

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