A NEW NEUROHAEMAL ORGAN IN ODOIPORUS LONGICOLLIS (OLIV.) (COLEOPTERA: CURCULIONIDAE)

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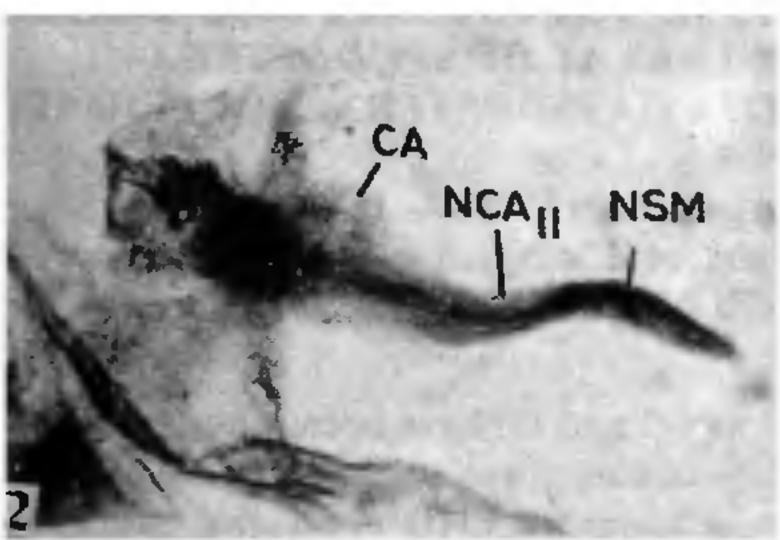
CORPUS cardiacum, dorsal aorta and corpus allatum are the three principal neurohaemal organs for cerebral neurosecretion in insects. Besides these, there may be numerous other organs which store and release neurosecretory material. The perisympathetic neurohaemal organs, lateral cardiac nerves, neuropilar neurohaemal organs, hypocerebral ganglion and oesophageal nerves may act as associated neurohaemal organs in different insects¹. Except in Gryllidae²⁻⁵, the nervi corporis allati-II has not so far been reported in any other family. The present investigation deals with the presence of nervi corporis allati-II in the curculionid weevil. Odoiporus longicollis.

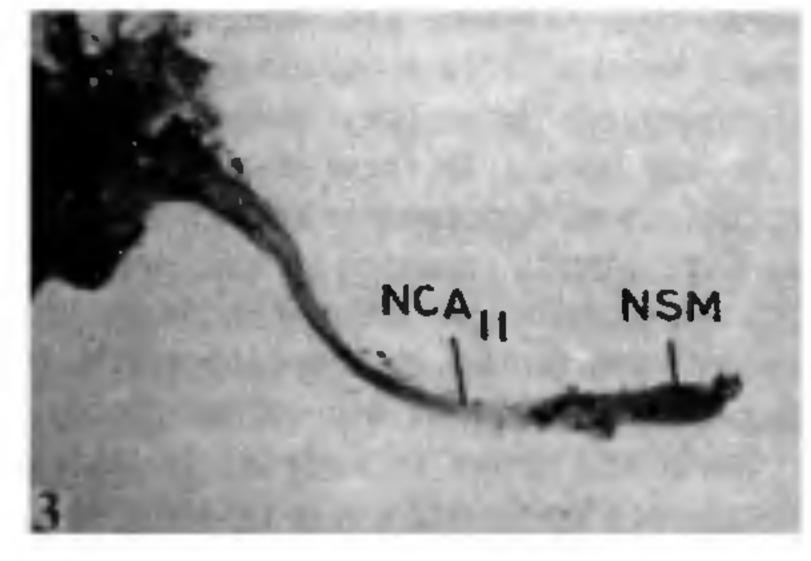
Adult O. longicollis of both the sexes and mixed age group were collected from local banana gardens. The retrocerebral endocrine complex was dissected in saline and fixed in aqueous Boum's solution. It was stained with paraldehyde fuchsin for in situ demonstration⁶.

The paired corpora allata are somewhat spherical egg-shaped glands, attached with the corpora cardiaca of their side by nervi corporis allati-I (NCA I) (figure 1). The axons of NCA I, either after passing directly over the surface of the corpus allatum middorsally or after spreading over the surface of the corpus allatum from dorsal to ventral forming a sort of network, emerge from the corpus allatum as the nervi corporis allati-II (NCA II) (figure 2). At the point of emergence of NCAII accumulation of neurosecretory material can be seen. The terminal end of NCA II dilates forming a sort of saccular structure (figure 3) which contains much neurosecretory material. The material is similar to that found in corpora cardiaca, which strongly suggests that the saccular part of the NCA II serves as a storage organ for cerebral neurosecretion.

Fate of NCA II as neurohaemal organ has been reported only in Gryllidae¹. The other workers^{2,5} observed the attachment of NCA II to the suboesophageal ganglion and number of neurosecretory axons passing through the NCA II reach the suboesphageal ganglion. But in O. longicollis the NCA II does not join with the suboesophageal ganglion. The saccular terminal end of NCA II lies freely in the haemocoel in the prothoracic region. The fibres of the NCC passing through the NCA II end in its saccular region, which is heavily lodged with neurosecretory







Figures 1-3 In situ demonstration of the retrocerebral endocrine complex with paraldehyde fuchsin showing corpus cardiacum (cc), corpus allatum (CA), nervi corporis allati I and II (NCA I, NCA II). 2. Emergence of nervi corporis allati II (NCA II) from corpus allatum (CA) (PF, in situ). NSM = Neurosecretory material. 3. In situ demonstration of the nervi corporis allati II (NCA II) having neurosecretory material (NSM) in the swollen terminal end (PI).

material. Such type of arrangement has not been reported so far. Thus, it is concluded that possibly the saccular part of NCA II releases neurosceretory material through its walls directly in the haemocoel.

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EFFECTS OF PRECOCENCE II ON LAST INSTAR LARVAE OF SPODOPTERA MAURITIA (LEPIDOPTERA: NOCTUIDAE)

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PRECOCENES (I and II) have been reported to inhibit the function of corpora allata, and consequently affect the juvenile hormone (JH) controlled processes of development, like moulting and metamorphosis in several insect species¹⁻⁵. Such morphogenetic effects of these compounds have been noticed mostly in hemimetabolous insects. In these insects precocenes induce precocious metamorphosis as well as a delay in moulting. Experiments reported in this paper were designed to test whether treatment of last instar larvae of Spodoptera mauritia Boisd. (Lepidoptera: Noctuidae) with precocene II (PII) will induce precocious metamorphosis and delay in ecdysis. In addition, experiments were also carried out to find out whether treatment of juvenile hormone analogue (JHA) would reverse the effects caused by the application of PII.

Last instar (6th instar) larvae of S. mauritia of different age groups, e.g. freshly ecdysed, or 0-day old, 1-day and 2-day old, were obtained from laboratory stock culture⁶. These were treated with PII either as a single dose on 0-day or as repeated daily doses from 0-day till pupation. PII (gift from Prof. W. S. Bowers, New York State Agriculture Experimental Station, Geneva, N.Y.) was dissolved and diluted in acetone so as to obtain 100, 80 and 60 μ g/ 5μ l. Larvae treated with an equivalent volume (5μ l/larvae) of acetone in a similar manner were kept as controls. JHA, ZR-512 (gift from Dr. S. Siddall, Zoecon Corp., Palo Alto, California, U.S.A.) was also diluted in acetone to get 0.2μ g/ μ l. 6th instar larvae were treated with PII on 0-day and 1-day followed by a dose of JHA on 2-day.

PII or JHA was applied topically on the larvae using a Hamilton microliter syringe.

Treatment of 6th instar larvae of S. mauritia with single or repeated daily doses of PII failed to induce precocious adult development but prolonged larval-pupal period (table 1). Larvae treated with PII pupated in 7-8 days whereas control larvae pupated in 5 days. Repeated application of PII produced various abnormalities in the ecdysed animals. In most cases they failed to shed their larval cuticle and retained larval legs (Figure 1 A,C). Pupal case partially covered the abdominal region. Further, when PII pretreated animals were given a single dose of JHA, the larvae pupated in 5 days and the pupae appeared normal (table 1).

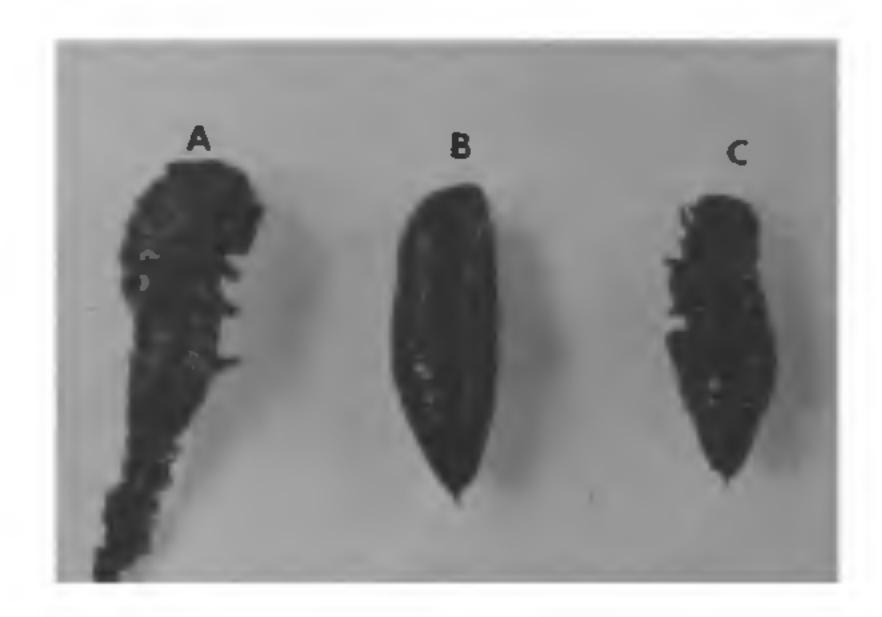


Figure 1. Abnormal pupae (A & C) induced by repeated topical application of 60 μ g precocene II to 6th instar larvae of S. mauritia. B. Normal pupa.

The experiments show that treatment of the last instar larvae of S. mauritia with PII prolongs larvalpupal period. In S. mauritia it is suggested that the delay in pupation is caused by the decreased titre of JH in the haemolymph, because JHA application to PII pretreated larvae reduced the larval-pupal period to its normal duration. A similar phenomenon was observed in Mamestra brassicae where allatectomy caused a significant prolongation of larval-pupal period⁷. It has also been reported that in lepidopterous insects, in addition to the activation by prothoracotropic hormone, an increase in JH titer in the prepupal state is necessary to induce prothoracic glands to their maximal rate of secretion 7,8. In view of these findings it seems that in S. mauritia, because of the decreased titre of JH in the haemolymph, the prothoracic glands secrete ecdysone at a slower rate delaying pupation. The production of abnormal pupae as a result of repeated application of Pl1 on 6th instar larvae seems to indicate that at high doses PII retards progressive adult development. Juvenilizing property