cally active DNA of the chromosome conforms to 'the original type' of the eutherian mammals.

The meiotic events are similar to the house shrew, *Suncus murinus*[5]. The sex vesicle resolves into a positively heteropycnotic bivalent in pachytene and becomes prominent in the later stages. In diplotene, the *X* and *Y* chromosomes show a distinct chiasma. The chiasma formation appears to be between the long arm of the *X* and *Y* chromosome (figures 4 and 4a). In diakinesis, the sex bivalent exhibits end-to-end association with the terminalization of chiasma (figure 5). Clear chiasma between the *X* and *Y* chromosomes, is a rare feature among eutherian mammals, suggesting the presence of homologous segments between them[5–7]. Occurrence of a distinct chiasma in *S. etruscus* adds one more instance to the list in mammals.

The present karyotype seems to be an usual karyotype of *S. etruscus* as it is consistent in number and structure in all the individuals studied. The *X* chromosome larger than 'the original type' and a clear chiasma between the sex chromosomes in both the species of *Suncus* appear to be implicated in the karyotype evolution of this genus.

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JUVENOMIMETIC EFFECTS OF SOLASODINE ON *CHILO PARTELLUS*

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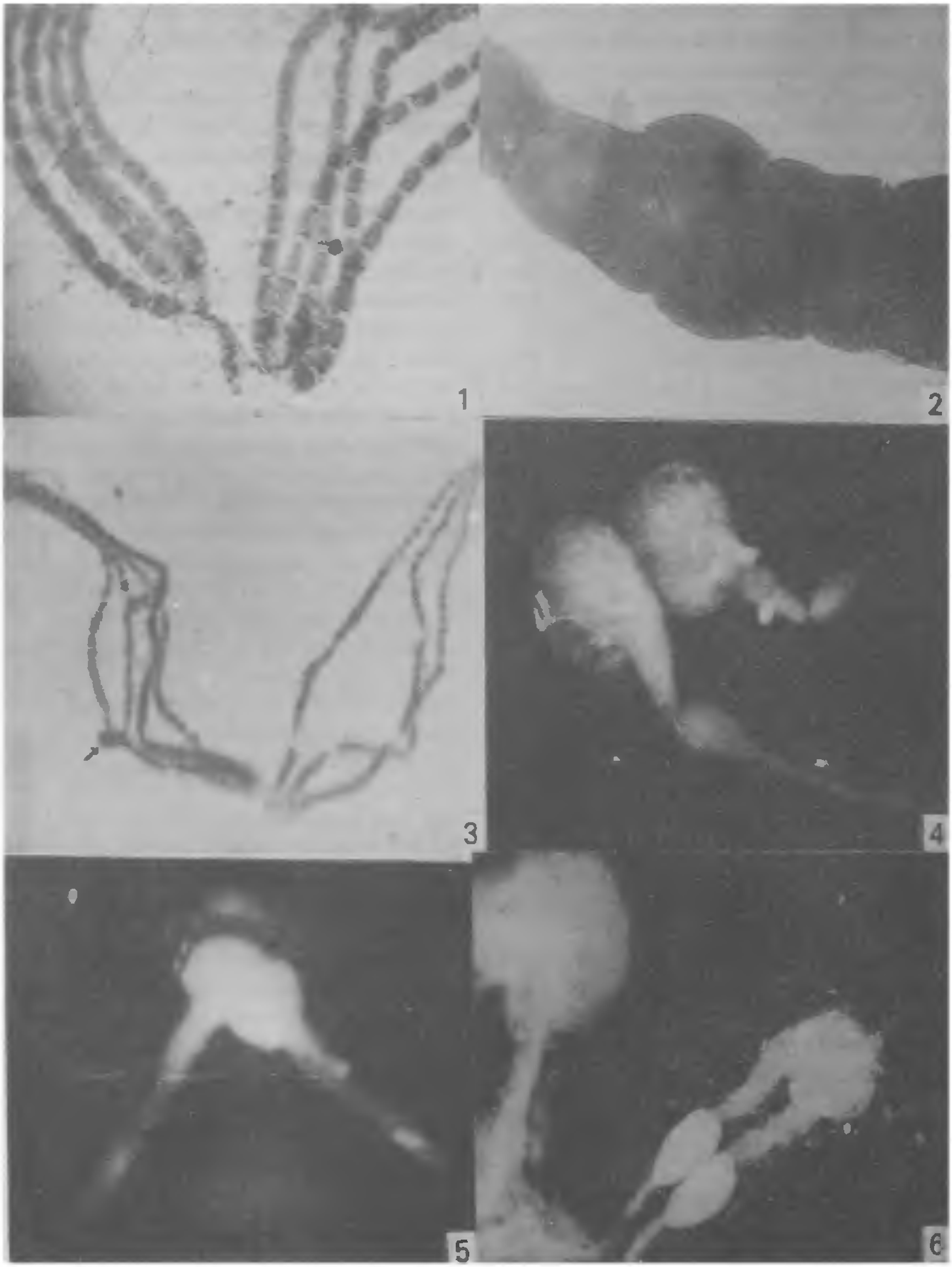
THE responses of insects to various juvenile hormone analogues derived from plants have been studied previously by different workers[1–4]. In this communication we report the juvenomimetic effects of solasodine[5,6] extracted from the green fruits of *Solanum aviculare* on the jowar stem borer *Chilo partellus* Swinhoe (Lepidoptera: Pyralidae).

The jowar stem borer *Chilo partellus* was reared on artificial diet[7] at a temperature of 27 ± 1°C and RH 65 ± 5%. Freshly ecdysed fifth instar larvae were treated topically on the abdominal region with 5 μls of different concentrations of solasodine (0.25–1 μg/μl) in acetone. Thirty larvae were treated (1.25, 2.5 and 5 μg/larva) each time and the experiments were replicated five times. Controls were treated with an equivalent volume of carrier solvent acetone. After total absorption of solasodine the larvae were transferred into the artificial diet and were observed daily to note the changes.

The duration of the last larval instar in controls usually varied between 11 and 13 days which, however, extended during the diapause state of the borer larvae[8]. Application of solasodine caused morphogenetic aberrations which were expressed in the next moulted forms.

At 5 μg/larva dosage the larval life prolonged by 5–7 days to that of control. Eighty-two per cent of the larval-pupal intermediates showed more larval characters, their length was reduced, the body was shrunken, the abdominal legs were absent but prolegs were present with their tips chitinized. Such forms were inactive and could not spin their cocoons and soon died. While at 2.5 μg/larva 86% of the larvae moulted after 15–16 days of treatment into larval-pupal intermediates with more pupal characters showing head, thorax larval and abdomen pupal and these intermediate forms died after 2–3 days. At lower concentrations of 1.25 μg/larva the larvae moulted into normal pupae and externally normal adults eclosed from these pupae.

The resultant externally normal female adults were dissected in insect Ringer solution. The ovariole length differed between the members of the same ovary or of both the ovaries. The number of



Figures 1–6. 1. Asymmetric ovary having 5 ovarioles. **2.** Ovariole showing compound egg chamber (×60). **3.** Ovary showing fusion between two points of an ovariole forming loops (arrow). **4.** Two developing testes unfused(×60). **5 & 6.** Imperfect fusion of two testes (arrow) (× 60).

ovarioles in one of the ovaries increased by 1 to that of control (figure 1). Frequently several oocytes were enclosed in a single long tube-like chamber without clear demarkation into individual oocytes (figure 2). Loops developed in some ovarioles (figure 3).

The resultant externally normal male adults were dissected. In a few cases the two developing testes did not fuse into a single sac, they remained wide apart and formed two separate testes each with separate vas deferens and seminal vesicle (figure 4). In other cases the fusion was incomplete and a constriction persisted (figures 5, 6). In controls the two testes were enclosed in a single sac. Such morphological deformities in the testes have been reported earlier with hydroprene[9].

The present finding therefore indicates the importance of solasodine as an anti fertility agent for *C. partellus*.

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ROOT-KNOT NEMATODE, *MELOIDOGYNE JAVANICA* BREAKS WILT RESISTANCE IN CHICKPEA VARIETY 'AVRODHI'

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A WILT resistant chickpea variety 'Avrodhi' was found infected with root-knot nematode, *Meloidogyne javanica* and *Fusarium oxysporum* f. sp. *Ciceri* at the university research farm. Root-knot nematodes are known for their ability to incite marked anatomical changes in their hosts. There are reports that infestation by root-knot nematodes increases susceptibility for pathogenic fungi and bacteria[1]. A histopathological observation was, therefore, undertaken to determine this aspect. Five surface-sterilized seeds of chickpea variety 'Avrodhi' were sown in pots containing autoclaved sand and soil (1:1) mixture. The fungus was cultured on potato dextrose broth (50 ml contained in 250 ml conical flasks) and the fungal mat was blended before inoculation. Fungal suspension (50 ml) and 500 larvae of *M. javanica* were used as inoculum. The treatments were: (i) fungus alone, (ii) nematode alone, (iii) fungus + nematode simultaneously, (iv) fungus 10 days prior to nematode, (v) nematode 10 days prior to fungus, and (vi) check (uninoculated).

One-week-old seedlings of chickpea were thus inoculated and after appearance of wilt symptoms, inoculated and uninoculated roots were collected. washed and fixed in F.A.A. for histopathological studies[2].

The plants, inoculated with fungus alone exhibited slight wilt but wilting was maximum and rapid in the plants where nematode preceded the fungus. Histologically roots, inoculated with nematode alone or in combination with the fungus, showed typical giant cell formation, development of egg masses, larvae and female nematode inside the roots (figures 1 and 2). This provided the fungus an easier access for entrance and rapid development of the disease (figure 3), with the nematode as a primary pathogen and the fungus as a secondary pathogen. Apparently, nematode infestation predisposed the host by changing its physiology and rendering it more susceptible to fusarium wilt damage. The results add to the understanding that due to change in physiology of the host, wilt fungus was able to break the host resistance.