itself was characterized by a steady rate of synthesis of 143 pg gland^{-1} h^{-1}.

When the onset of receptivity appeared at the beginning of vitellogenesis (72–83 h), rates of JH synthesis/release in receptive and unreceptive females were not significantly different (approximately 200 pg gland^{-1}h^{-1}). However, at 92 h, the rate of JH III synthesis/release by CA from receptive females increased significantly, reaching a rate of 533 pg gland^{-1} h^{-1} at chorionation. By contrast, synthesis/release by CA from unreceptive females did not increase significantly during this same period of time.

Discussion. The results of the present study show that JH III is the sole JH in *C. vomitoria,* in agreement with previous identifications of the JH in Diptera[8–11]. The measurement of the biosynthesis/release of JH III by *Calliphora* CA in vitro is the first such demonstration for dipteran CA, and the developmental study of gland activity during the gonadotropic cycle indicated a coincidence of increasing gland activity with the onset of receptivity. The nature of the relationship between receptivity, JH and ovarian development has yet to be resolved, although some information is available from a previous study[4]. On the basis of the results of that study, it was concluded that the occurrence of receptivity coincided with the beginning of vitellogenesis and that JH was necessary for receptivity. This latter conclusion was derived from the fact that vitellogenesis and receptivity were abolished in allatectomized females and that the application of JH analogue to the allatectomized animals restored both processes. Furthermore, the ovary itself appeared to have a role in the maintenance of receptivity, since less than 24% of ovariectomized females remained receptive[4]. It could be postulated from this information that the JH titer stimulates vitellogenesis in *C. vomitoria,* as it does in other fly species[5–7]. The occurrence of receptivity may then be affected indirectly by JH, through the ovarian development resulting from vitellogenesis. However, it is also possible that JH affects receptivity directly. Supporting this possibility it was shown, in this work, that ovarian development is the same in both receptive and unreceptive females and that receptive females possess a higher JH titer than unreceptive females. The function of the ovary may thus be to maintain receptivity once established, rather than to affect its initiation.

There also appears to be a link between receptivity and ecdysteroid levels. In *C. vomitoria,* it has been found that ecdysteroid levels are highest just at the onset of vitellogenesis and receptivity[3,4]; the same situation appears to occur in *Musca domestica*[14,15]. Thus the *Calliphora* system may prove to be ideal for studying the interendocrine relationships of JH and ecdysteroids in the regulation of a reproductive process. In this species, critical questions remain to be answered concerning the role of JH and ecdysteroids in the regulation of vitellogenesis, ovarian development and receptivity. For example, does JH directly or indirectly affect receptivity? What is the specific role of the ovary in this process? With the in vitro systems currently available, solutions to these problems should be forthcoming.

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Ecdysteroid-dependent larval-adult oviduct transformation in the milkweed bug *Oncopeltus fasciatus* requires absence of juvenile hormone[1]

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Summary. It has been tested whether juvenile hormone plays a role in the larval-adult transformation of lateral oviducts in the milkweed bug. The transformation is ecdysteroid-dependent, as was reported previously[2]. Application of precocene or juvenile hormone III proved that the absence of juvenile hormone is required.

Key words. Insect oviduct; larval-adult transformation; juvenile hormone; *Oncopeltus.*

The lateral oviducts of the milkweed bug *Oncopeltus fasciatus* undergo a drastic larval-adult transformation during the last larval instar. Between day 3 and day 5 the long and thin larval oviducts shorten and become very wide. Previous studies have shown that this process is ecdysteroid-dependent in a dose-related manner[2]. Juvenile hormone is expected to be absent in the stage preceding adult molt[3]. We therefore examined whether the oviductal transformation requires the absence of juvenile hormone or not.

Materials and methods. Oncopeltus fasciatus Dallas (Heteroptera, Lygaeidae) was reared on peeled sunflower seeds at 27.5 °C, in a relative humidity between 60% and 70% and without photoperiod. The animals were staged at 4th, 5th (= last larval) or adult molt. Those which ecdyzed during the night were separated the following morning and defined as day-0 adults, or as day-0 larvae of the ultimate (5th) or penultimate (4th) stage.

The treatment with precocene II (Aldrich, Steinheim) was

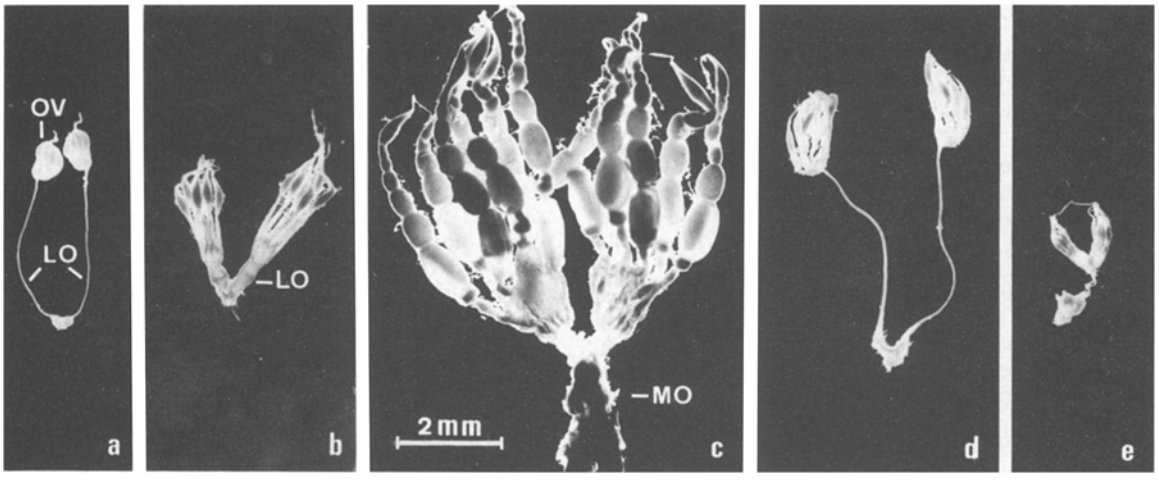

Figure 1. The female genital tract at various developmental stages and after experimental manipulations; LO: lateral oviduct, MO: median oviduct, OV: ovary; the magnification is the same in all micrographs and is as indicated in (c). *a* Normal genital tract prior to larval-adult transformation (5th larval instar day 2). *b* Normal genital tract after larval-adult transformation (5th larval instar day 5). *c* Normal mature genital tract (imago day 10). *d* Genital tract of juvenile hormone-induced supernumerary larva ('6th' larval instar day 1). *e* Genital tract of a precocene-induced precocious adult (day 2 after precocious adult molt).

performed on day-0 4th instar larvae. 2 µl of an acetone solution containing 20 µg of the anti-juvenile hormone[4] precocene II were applied topically to the abdominal dorsum. The treatment with juvenile hormone III[5] (Sigma, Deisenhofen) was performed on day-0 5th instar larvae. 2.5 µl of an acetone solution containing 25 µg of the hormone was applied in the same way as precocene II. The ecdysteroid titer of last instar larvae was determined by radioimmunoassay as reported earlier[6].

For morphogenetic studies on the female genital tract larvae were dissected in Grace's medium (Gibco, Karlsruhe), fixed and stored in Bouin's fluid. Outlines of ovaries, lateral oviducts and part of the median oviduct[7] were drawn with a drawing mirror and dissecting microscope from Wild (Heerbrugg). The length of the lateral oviducts was measured from the posterior end of the pedicels to the beginning of the median oviduct, and the index length/width (L/W) was computed. Light micrographs were taken through a Wild dissecting microscope equipped with a camera (Wild MP 45, MPS 51S).

Results and discussion. Normal oviductal development during 5th larval instar: larval-adult transformation. The proportions of the genital ducts change drastically during this larval instar (fig. 1). From day 3 to day 4 the index L/W (length/width) of the oviducts drops from 79 to 8 and further to 2 until adult molt; in imagines it is around 1 permanently (fig. 2). The transformation is triggered by an increase in the ecdysteroid titer (fig. 2), which was shown previously[2]. Since juvenile hormone is supposedly at a low level or not present in last instar larvae, the question arises whether its absence (or low titer) is mandatory or not. In a first experiment the anti-juvenile hormone precocene was applied to newly hatched 4th instar larvae to induce premature adult development and possibly premature oviductal transformation.

Oviducts in precocene-induced precocious adult females. The treatment of 4th instar larvae with precocene is followed by a precocious adult molt instead of a last larval one. It is known that precocene damages active corpora allata in *Oncopeltus fasciatus,* resulting in the failure of the gland to synthesize juvenile hormone[4, 8, 9]. The precocious adult development observed here is therefore interpreted as a consequence of the absence of juvenile hormone. The ecdysteroid titer is apparently not impaired by precocene, since molting processes take place. The genital system of day-2 precocious adults is small, but the oviducts have clearly undergone adult transformation (fig. 1). The index L/W is 0.9 as in normal imagines, and is quite different from that in 5th instar larvae of corresponding age, where it is 94 (fig. 2). This experiment indicates that it is the absence of juvenile hormone which allows the oviductal transformation at high ecdysteroid levels. And it is also clear that already at the penultimate stage, the genital ducts are capable of responding to metamorphic hormonal stimuli. In this respect they behave like other organs and structures which are subject to morphogenetic reorganization or accelerated growth during adult metamorphosis, e.g. epidermis, imaginal discs, salivary glands (Diptera), fat body (Diptera), brain (Lepidoptera), prothoracic glands (see Koolman and Spindler[10], for review), external sex organs[7]. Further evidence that the ecdysteroid-dependent transformation of oviducts requires the absence of juvenile hormone is provided by a second experiment described below.

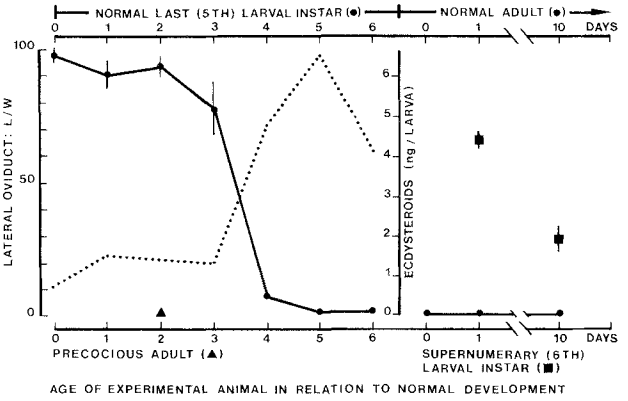

Figure 2. Morphometric alterations of the lateral oviducts expressed by the index L/W (length/width) during normal development of 5th larval instar and adult maturation (●—●) and in response to precocene and juvenile hormone treatment. A drastic decline of the index is seen between day 3 and day 4 of normal larval development. It correlates with the rise of the ecdysteroid titer: stippled curve (data from Dorn et al.[6]). Precocene-induced precocious adults contain transformed lateral oviducts (▲). Juvenile hormone-induced supernumerary larvae contain at day 1 non-transformed, larval lateral oviducts, whereas a partial transformation has taken place at day 10 (■). n = 10 for each datum point (±SEM); where no SEM is given, it is smaller than the datum point.

Oviducts in juvenile hormone-induced supernumerary larvae. Last instar larvae treated with juvenile hormone at day 0 undergo another (6th) larval molt leading to a supernumerary, giant larvae. The genital tracts of these larvae were dissected at day 1 and at day 10 (fig. 1). At day 1 the oviducts have definitely a larval form with an index L/W of 64 (fig. 2). At day 10 the index is 30, indicating an incomplete transformation. It is noteworthy that these animals have synthesized a new cuticle, but are unable to shed the old one. The new cuticle exhibits some adult characteristics. Its deposition testifies to the build-up of an ecdysteroid titer in supernumerary larvae. The incomplete oviductal transformation may be caused by persisting exogenous juvenile hormone, a (slight) activity of the corpora allata, a suboptimal ecdysteroid titer, a combination of these or still other factors.

The experiments have demonstrated unequivocally that transformation of the oviducts is not strictly linked to a developmental stage, i.e. 5th larval instar, but is regulated by specific hormonal signals: It is evoked by high ecdysteroid titer, but requires the absence of juvenile hormone. If these conditions are created prematurely (by precocene), or if they are postponed (by juvenile hormone treatment), oviductal transformation can be shifted to earlier or later stages in development. This is comparable with the hormonal regulation of larval-adult transformation of other tissues and organs[10].

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Binding of (^{125}I) triiodothyronine to human peripheral leukocytes and its internalization

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Summary. Ultrastructural autoradiography showed high specific binding of (^{125}I) triiodothyronine, as confirmed by a competition test, to plasma membranes, nuclei and mitochondria of human peripheral leukocytes. A high level of binding was also noted on the granulocytes' granules, especially in eosinophils.
Key words. Triiodothyronine; binding sites; ultrastructural autoradiography.

The binding of triiodothyronine (T_3) has been biochemically demonstrated in plasma membrane[1], cytosol[2], mitochondria[3] and nuclei[4], usually of rat hepatocytes. Autoradiographic studies confirmed T_3 localization in the nuclei and mitochondria, but not on the plasma membrane; clear-cut results regarding T_3 binding to different organelles were not obtained, probably due to differences in the methods and cell types used (e.g. hepatocytes[5-7], lymphocytes[8], lung alveolar cells[9], posterior hypophysis and median eminence cells[10]). We have used ultrastructural autoradiography to investigate T_3 binding and its intracellular distribution in human peripheral leukocytes, cells which can be easily obtained without damaging manipulations.

Material and methods. Human leukocytes from heparinized peripheral blood of euthyroid donors were obtained after 60 min of sedimentation at room temperature and 3 washings in Eagle's minimal essential medium (MEM) for 10 min at 200 × g. Two parallel sets of samples containing 15×10^6 leukocytes were incubated for 30 and 60 min at 37 °C in 1 ml either of MEM with 10^5 Bq (^{125}I) T_3 (sp. act. 44 TBq/g) or under the same conditions with a 50–100-fold excess of unlabeled T_3. Lymphocytes isolated according to Bøyum[11] were incubated under the same conditions as the leukocytes; control lymphocytes with a 200-fold excess of unlabeled T_3. All samples were washed 3 times for 10 min at 200 × g in MEM cooled to 4 °C, and fixed in 4% paraformaldehyde. Ultrathin sections from pellets embedded in Epon-Araldite were prepared by standard methods. The stability of T_3 binding was proved in a special set of samples. Autoradiograms were prepared after Williams[12a] using Ilford L4 emulsion, and developed physically and chemically. 103 experimental and 93 control autoradiograms of isolated lymphocytes were evaluated morphometrically by the circle method[12b] at a final magnification of 25,000 times.

Results. Autoradiograms of leukocytes exhibited binding on the plasma membrane (PM), mitochondria (M), tubulovesicular structures (TV), granules (G), lysosomes (L) and nuclei (N). In the eosinophilic granulocytes, a high level of binding appeared on eosinophilic granules (EG) (fig. 1). Control leukocytes displayed lower labeling than experimental cells.

In isolated lymphocytes, the binding was analogous to that of the leukocytes, i.e. over the plasma membrane, the cytoplasm and the nuclei (fig. 2). Morphometric analysis of the samples after 60 min of incubation proved non-random distribution of grains and specific binding in mitochondria, plasma membrane and nuclei. The intensity of the labeling of cell structures was as follows: M > PM > N > C/N border > other cytoplasmic structures (C).

Control lymphocytes displayed a lower labeling and morphometric analysis revealed another sequence of labeling intensity: C > M > C/N border > N > PM (table).