0.001% emulsion of the IGR was completely stopped. Many larvae died already in the first ecdysis, and those of the male larvae, which passed through, proceeded successfully only to the nymphal stage but failed to metamorphose into adults.

The affected nymphs died mostly in ecdysis but some of them succeeded actually in moulting into intermediary forms between the supernumerary nymphs and adults (intermediates I) characterized by nymphal-like short wing buds and adult-like anal plates at the abdominal tip covered with pores (figure 3). The intermediates started 1–2 additional ecdyses which were never finished with eclosion. The lactophenol whole mounts often revealed, underneath the skin of the intermediates I, 1–2 cuticular layers of intermediates II and III. Thus the morphogenetic changes occurring during 2–3 supernumerary instars could be studied on the same animal (figure 4). The most striking feature was the reduction in the number of pores on the anal plates in the succession from the intermediate I (uppermost skin) with about half the number (35–50) to the intermediate III (the very inner skin) with only few pores left (0–3). Simultaneously, also the tip of the abdomen is getting more blunt in each successive stage.

The anal pore plates of the male mealy bugs are the adult characteristic which differentiates during the nymphal metamorphosis instar. The reduction of the number of these pores in 2–3 successive nymphal-adult intermediary instars is a new example of reversal of an adult structure induced by a JH-active substance in an intact insect. This observation is consistent with the results of experiments which proved that the adult epidermis has the capacity to secrete juvenile cuticle when transplanted into or connected with the larval milieu[6–11]. JH appears to be responsible for these changes as JH-active IGRs applied to *Tenebrio* pupae cause a 'larvalization' of the cuticle[12]. All these results prove that JH and JH-active IGRs possess an intrinsic morphogenetic activity which is exerted directly on the target tissues. Many parts of the epidermis remain capable of reverting from the more differentiated type back to the larval pattern under the influence of JH; the other parts are early committed irrevocably to lay down imaginal structures[13].

The JH-induced reversal of metamorphosis can be well explained by an activation of specific genes of conservative characteristics[14]. Another concept which views the JH as a repressor of the new genetic information, the status quo concept[15,16], leaves no room for any such reversion of the once attained degree of differentiation. Apart from its direct morphogenetic effect, JH seems to influence the metamorphosis also indirectly by reducing the titre of the moulting and differentiation hormone ecdysone in the blood[17]. This dual role of JH explains the inhibition of ecdysis occurring parallel to the morphogenetic changes in mealy bugs and many other insects treated with JH-active IGRs. On the other hand, the ecdysone inhibiting role of JH, separated from the morphogenetic activity, is demonstrated by the death of the young larvae in the first ecdysis in mealy bugs[3], scale insects[18,19], roaches[20] and other insects[21] treated with JH-active IGRs.

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Juvenile hormone: Evidence for a role in the feeding rhythm of *Oncopeltus fasciatus* (Dallas)

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Summary. The diurnal feeding rhythm of female milkweed bugs was damped when fed nonhost seeds. Juvenoid treatment partially restored the normal rhythm. Precocene II-treated milkweed seed-fed females fed arrhythmicly during the light phase without reduced total feeding activity. This effect was largely prevented by simultaneous treatment with JH III.

Little is known about the role of hormones in insect circadian rhythms. Possible neuroendocrine coupling in the circadian locomotor rhythms of a cockroach[2] and a cricket[3], as well as for spermatophore production in crickets[4] has been proposed. However, it has been pointed out[5,6] that the evidence is in no instance entirely consistent nor complete and hence needs further study. Truman and Riddiford's[7] demonstration of the involvement of a diffusable brain hormone in the circadian eclosion behaviour of silkmoths thus stands as the only undisputed example of involvement of a hormone in an insect behavioural circadian rhythm.

During the first week after eclosion, adult females of the large milkweed bug, *Oncopeltus fasciatus* (Dallas), gradually

1 I thank Dr Judith H. Willis, Univ. of Illinois, for providing facilities, Meyer Schwartz of ARS, USDA, Beltsville, MD, USA, for providing the precocene II, and Dr W. S. Bowers, formerly of ARS, USDA, Beltsville, MD, for supplying the juvenoid. This work was supported in part by grant AG–00248 from the National Institutes of Health to J. H. Willis.

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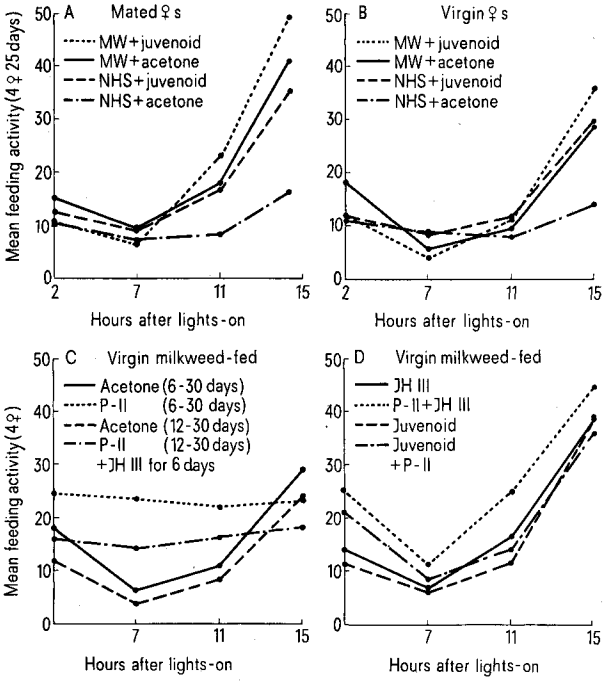
Standard errors for data in the figure and total feeding activity

Treatment*	Standard errors 1 h	7 h	11 h	15 h	Total feeding** activity	SE
NHS, M, A	0.7	0.7	0.7	1.0	43.0	2.3
NHS, M, J	1.2	1.0	1.4	1.7	73.6	3.7
MW, M, A	2.1	1.6	2.7	2.6	83.6	4.2
MW, M, J	1.9	1.6	2.0	1.9	89.7	4.4
NHS, V, A	0.7	0.8	0.7	0.8	41.2	2.5
NHS, V, J	1.0	0.7	0.5	2.1	62.0	2.6
MW, V, A	1.3	1.9	1.3	2.7	62.0	2.9
MW, V, J	1.6	1.9	1.8	2.0	62.9	2.2
MW, V, A	3.1	1.9	1.8	2.1	64.0	6.3
MW, V, J	1.3	1.3	2.1	1.9	67.5	0.5
MW, V, JH III	2.1	1.4	2.7	2.9	78.0	4.9
MW, V, P-II	3.2	4.2	3.6	4.9	94.4	8.7
MW, V, J, P-II	2.1	1.9	2.4	1.7	80.5	6.4
MW, V, JH III, P-II	4.2	2.5	2.3	5.0	106.1	9.8
MW, V, A (12–30 days)	1.2	0.8	1.5	2.8	48.8	5.5
MW, V, P-II (12–30 days)	3.2	0.9	2.9	1.6	65.3	9.4

* NHS, nonhost diets; MW, milkweed seed diet; M, mated; V virgin; A, acetone; JH, juvenile hormone; J, juvenoid; P-II, precocene II.
** The sum of the mean activity for each time period for the given observation period. The potential maximum for the 6–30-day-period is 400 and for the 12–30-day-period is 304.



Effect of diet, juvenoids and precocene II on the diurnal feeding rhythm of female milkweed bugs under a 16L-8D photoperiod. 2 µg of JH III or the juvenoid or 10 µg of precocene II were topically applied every other day to females in certain treatments. Precocene II treatments began the day before eclosion and juvenoid or JH III treatments began the day of eclosion. The values given are the means of the sums of feeding activity for each replicate consisting of 4 females for the given observation period. The potential maximum value for the 6–30-day-period is 100 and for the 12–30-day-period is 76.

develop a distinct diurnal feeding rhythm which may be circadian, although it decays within 1–3 days under constant light or darkness[8]. I tested the possibility that juvenile hormone (JH) is a regulatory component of this rhythm by inducing a partial or complete JH deficiency by a) treating adult females with the antiallatotropic agent precocene II[9] and b) feeding females nonhost seeds[10], and then attempting to prevent the observed effects by simultaneous treatment with a juvenoid.

Methods. Adults for all experiments were obtained from nymphs reared at 25 °C on milkweed seeds. In experiment 1, groups of 4 virgin or mated females were fed milkweed seeds or 1 of 3 nonhost seeds: sunflower, cashew or walnut. Some groups on each diet were topically treated with 2 µg of the juvenoid (E)-4-[(6,7-epoxy-3-ethyl-7-methyl-2-nonenyl) oxy]-1,2-(methylenedioxy) benzene[11] every other day. Feeding activity, defined as the number of bugs with the stylets anchored in a seed, was observed at 2, 7, 11 and 15 ± 1 h after lights-on in a 16L-8D cycle at 25 °C. The data for days 6–30 after eclosion were pooled to obtain the means for each treatment. The results for the 3 nonhost diets were similar enough to justify pooling the data for these diets for simplicity of presentation (figure A, B; table). In experiment 2 milkweed-fed virgin females were treated a) with precocene II, b) with both precocene II and the juvenoid or JH III, applied to different areas of the abdomen on alternate days, c) with precocene II throughout the 30-day period and with JH III for only the first 6 days. Otherwise the methodology was the same as in experiment 1.

Results. Damping of the rhythm as well as reduced total feeding activity (the sum of activity at 2, 7, 11 and 15 h) was observed on all nonhost diets in both virgin and mated females (figure A, B; table). Juvenoid treatment

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partially restored the rhythm on the nonhost diets, primarily by increasing feeding during the late photophase. Precocene II caused an arrhythmic feeding pattern (figure, C), without reducing total feeding activity (table), while simultaneous treatment with the juvenoid or JH III largely restored the normal rhythm (figure, D). Furthermore, when JH-III-treatment was discontinued after the 6th day, the rhythm was essentially eliminated between the 12th and the 30th day (figure, C), indicating that JH was not just involved in the initiation of the rhythm.

Discussion. One must consider the possibility that the feeding rhythm may be influenced by the oviposition rhythm, as the period of maximal oviposition coincides with the minimal feeding period during the photophase[8]. Since JH is also essential for ovarian maturation[12], JH deficiency might induce photophase feeding arrhythmicity indirectly by eliminating oviposition behaviour. However, a pronounced rhythmicity was evident in males and in virgin milkweed-fed females although they oviposited a mean of only 1.9 times during the 30-day-period. Also, as previously observed[8], cyclic feeding is initiated before oviposition begins, indicating that the 2 cycles are not obligatorily coupled.

Apparently, JH does not act as a proximal stimulus for development of the rhythm, as juvenoid treatment of young females did not cause its precocious appearance (unpublished data). Rhythm development is characterized by a reduction in feeding activity during times other than the peak evening hours which change relatively little[13]. This suggests a cyclic inhibitory influence is mainly responsible for the rhythmicity. This conclusion is in agreement with the marked increase in feeding activity during the afternoon (7 and 11 h) induced by precocene II treatment. Ironically, the results of the nonhost diet treatments, in themselves, suggest just the opposite: That the juvenoid accentuated rhythmicity primarily by stimulating peak evening feeding (figure, A). However, in view of the total evidence, the following interpretation seems more likely: The normal level of total feeding activity observed in precocene II-treated females (table) suggests that JH is not involved in the stimulatory effect of milkweed seeds on feeding activity. Apparently, juvenoids can largely or completely substitute for this specific seed effect in females when on nonhost diets, perhaps by some indirect means, such as stimulation of ovarian maturation. Thus, the more normal feeding activity pattern seen in juvenoid-treated females on nonhost diets appears to be due both to a general stimulatory effect on feeding and to the enhancement of a cyclic inhibitory influence.

It appears that the juvenoid treatments may not have completely prevented precocene II or dietary-induced rhythm damping, as in all 4 comparisons the 7/15 h feeding activity was lower than for the control juvenoid-treated females. This may well be due to a failure to restore a completely normal diurnal JH titre fluctuation.

While these experiments do not distinguish between a permissive vs. a regulatory role for JH in the feeding rhythm and do not exclude the possibility of an indirect mode of action, they do provide the first encouraging evidence that JH may be an essential component of some repetitive insect behavioural rhythms.

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Gastrin: Obligatory intermediate in the postprandial mobilization of gastric histamine in the rat[1]

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Summary. In unoperated fasted rats, feeding raised the serum gastrin concentration, reduced the gastric mucosal histamine content and activated the gastric histidine decarboxylase. The reduction of gastric histamine and activation of histidine decarboxylase was induced also by the injection of pentagastrin. In antrectomized rats, feeding failed to produce these effects. Injection of pentagastrin, however, still lowered gastric histamine and activated gastric histidine decarboxylase. Thus, antral gastrin seems to be an obligatory mediator of the postprandial activation of histidine decarboxylase and mobilization of histamine.

Histamine in the rat stomach is predominantly located in endocrine-like cells of the oxyntic mucosa[2-4]. Ultrastructurally, these cells comprise 2 types, distinguishable from each other by the morphology of their secretory granules[3,4]. These cell populations seem to respond to gastrin in the following 2 ways: 1. Gastrin mobilizes histamine and activates synthesis of the histamine-forming enzyme[4,5]. 2. Gastrin exerts trophic control of at least 1 of the 2 histamine-storing endocrine-like cell types[4].

Feeding after a period of fasting activates gastric histidine decarboxylase and elicits a marked but short-lasting reduction of the gastric histamine content[6]. This reduction in histamine content is thought to reflect mobilization of histamine[6]. It has been claimed that the effects of feeding on the histamine-storing cells are the result of direct vagal excitation in conjunction with the postprandial increase in the serum gastrin concentration[6,7].

In a series of studies, it has been demonstrated, however, that gastrin is the major intermediate in the activation of histidine decarboxylase, and that the contribution of

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