stored in cells of the pars tuberalis. Therefore, the LH content of the PT-ME region should in actuality be regarded as the LH content of the pars tuberalis.

The present results clearly indicate that the LH content of the pars tuberalis of androgenized female rats is much lower than that of control females at oestrus. On the other hand the LH contents of the pars distalis of androgenized rats and that of the pars distalis of control females at oestrus are not significantly different[11]. Therefore, under the same experimental conditions the LH cells of the pars tuberalis and the LH cells of the pars distalis behave in different way. Furthermore, the LH content of the pars distalis of male rats is much higher than that of the pars distalis of female rats at oestrus. On the other hand the LH content of the pars tuberalis of both males and females at oestrus is not significantly different (fig. 1). These latter results could be taken as an indication that under normal conditions the LH cells of the pars tuberalis and those of the pars distalis do not function as a homogeneous cell population. It is postulated that the LH cells of the pars tuberalis and those of the pars distalis should be regarded as 2 functionally different sources of LH.

It is worth noticing that the LH cells of the pars tuberalis of androgenized female rats do not behave like the LH cells of the male pars tuberalis as judged from the LH content of this pituitary region. A similar finding has been reported for the LH cells of the pars distalis of androgenized female rats[12].

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Why is *Pyrrhocoris apterus* insensitive to precocene II?[1]

M. Hodková and R. Socha

Institute of Entomology, Czechoslovak Academy of Sciences, Na Folimance 5, 12000 Praha 2 (Czechoslovakia), 12 February 1982

Summary. Precocene II (P II) was applied to the adult females of 2 Pyrrhocorid bugs - *Pyrrhocoris apterus* (insensitive to P II) and *Dysdercus cingulatus* (sensitive to P II) - subjected to allatectomy and intraspecific or interspecific reimplantations of corpus allatum (CA). The failure of P II to inhibit ovarian development in *P. apterus* appears to be caused by both a low sensitivity to P II of the CA itself and unknown 'anti-precocene mechanisms' outside the CA.

Precocene II (P II) has been shown to inhibit the ovarian development in several insect species[2]. The corpus allatum (CA) appears to be a target of P II as juvenile hormone analogues can compensate for the inhibitory effect of P II[2,3]. Histological evidence has been provided that P II selectively destroys the secretory cells of the CA[4-7]. The experiments in vitro suggest that P II inhibits the CA directly[8-10]. In vivo, it has been demonstrated in *Dysdercus cingulatus*[11] and *Oncopeltus fasciatus*[12] that P II inhibits the CA humorally rather than via the nervi allati. However, it is not clear whether the inhibitory effect of P II in vivo depends merely on the specific interaction between CA and P II or whether other physiological processes outside the CA are involved. A similar problem arises when we ask why the P II fails to inhibit ovarian development in *Pyrrhocoris apterus* (Socha, unpublished), although *D. cingulatus* from the same family of Pyrrhocoridae is highly sensitive to P II[2]. We have investigated this question in allatectomized females of *D. cingulatus* and *P. apterus* subjected to intraspecific and interspecific reimplantations of CA and to subsequent treatment with P II.

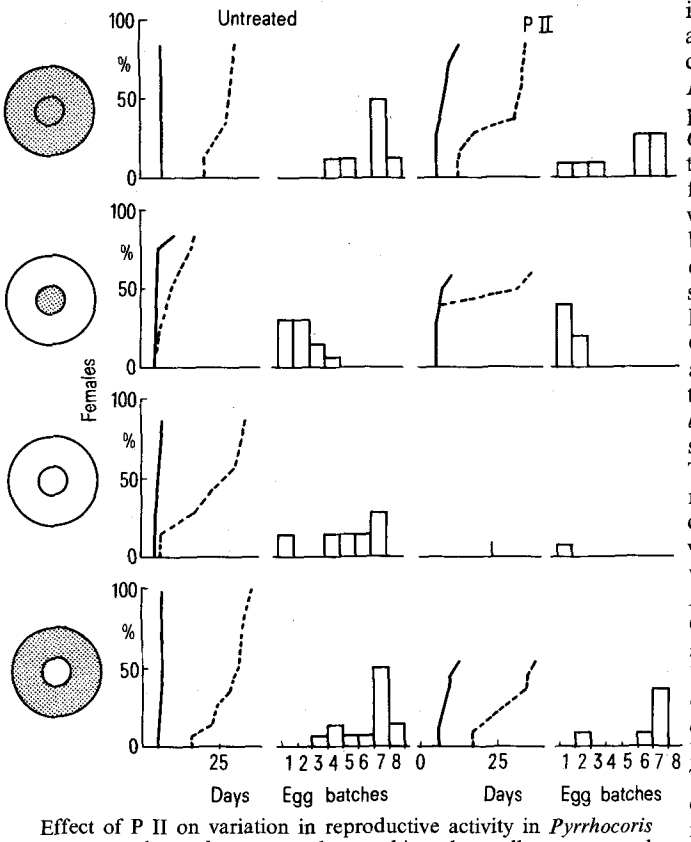
Materials and methods. Adult females of *P. apterus* and *D. cingulatus* (Heteroptera, Pyrrhocoridae) were used in the experiments. Insects were reared on linden-seed at 26 ± 1 °C and at long-day (18L:6D) photoperiod. The females destined to be donors or recipients of CA were deprived of food within several h after imaginal ecdysis. Next day the CA was excised from each recipient through an incission in

Table 1. Effect of P II on reproduction in unoperated *Pyrrhocoris apterus* and *Dysdercus cingulatus*

Species	n	Oviposited females (%)	Pre-oviposited period (days)	Oviposited period (days)	Post-oviposited period (days)	Egg batches/ female	Eggs/batch
*P. apterus**	9	100	6.4 (6-10)	22.8 (8-29)	2.3 (0-6)	6.9 (3-8)	55.2 (18-69)
P. apterus + P II*	9	100	7.8 (6-8)	21.0 (0-30)	4.4 (0-25)	6.0 (1-9)	57.5 (30-76)
*D. cingulatus***	10	100	9.7 (7-17)	13.4 (8-21)	6.9 (0-16)	4.1 (2-6)	77.1 (12-116)
D. cingulatus + P II**	8	0	-	-	-	-	-

Dose of P II per Petri dish: 600 µg. Experiments lasted 35 days* and 30 days**.

the neck membrane and one complex of CA + corpora cardiaca + aorta from another individual was immediately reimplanted. All surgical manipulations were performed under insect saline. The groups of females were transferred to Petri dishes (⌀ = 9 cm), coated with P II in the quantity indicated, within several h after the imaginal ecdysis (unoperated females) or after the operation. The females were isolated and paired with males 6 days later. The females which died within 1 week after the beginning of experiments were excluded. The inhibitory effect of P II was evaluated according to the suppression of the reproductive activity.



Effect of P II on variation in reproductive activity in *Pyrrhocoris apterus* and *Dysdercus cingulatus* subjected to allatectomy and intraspecific or interspecific reimplantations of corpus allatum. Solid line, cumulative incidence of females which started oviposition; broken line, cumulative incidence of females which ceased oviposition; large circles, recipient; small circles, complex of corpus allatum + corpora cardiaca + aorta; shaded, *Pyrrhocoris apterus*; open, *Dysdercus cingulatus*. Other information see table 2.

Results and discussion. Unoperated females. Table 1 shows that the reproduction of *P. apterus* females was not inhibited by P II, thus indicating that their CA remained active after P II-treatment. In contrast, P II inhibited the reproduction of all females of *D. cingulatus*. The sensitivity of *D. cingulatus* and the insensitivity of *P. apterus* in our experimental conditions has thus been confirmed.

Operated females. All results are summarized in table 2 and the figure. The reproductive parameters of *P. apterus* females with excised and reimplanted CA were similar whether the reproduction was induced by *P. apterus* CA or by *D. cingulatus* CA. The internal milieu of *P. apterus* is evidently equally suitable for the function of CA from both species. The internal milieu of *D. cingulatus*, on the other hand, is less favorable for *P. apterus* than for *D. cingulatus* CA as the fecundity of *D. cingulatus* females was lower after the interspecific than after the intraspecific reimplantation of CA. The incidence of oviposition start in *D. cingulatus* females was, however, similar without respect to the species of donor.

The treatment with P II was almost without effect on the reproduction in *P. apterus* females subjected to the intraspecific reimplantation of CA. In contrast, the reproduction was inhibited in almost 50% of allatectomized *P. apterus* which received *D. cingulatus* CA. It appears, therefore, that *D. cingulatus* CA is more sensitive to P II than *P. apterus* CA. This difference was even more evident when the allatectomized *D. cingulatus* females were recipients. While P II inhibited reproduction in almost all females reimplanted by *D. cingulatus* CA, only about 25% of females which obtained *P. apterus* CA had the reproduction impaired by P II.

The 'anti-precocene mechanisms' outside the CA became evident when the reproductive parameters of allatectomized recipients of the different species which were given CA from donors of the same species were compared. Thus, in the females which received *P. apterus* CA, P II decreased the incidence of oviposition by about 25% and the number of egg batches from 2.0 to 1.3 on average when the recipient was *D. cingulatus*, although there was almost no inhibition when the recipient was *P. apterus*. In the females

Table 2. Effect of P II on reproduction in *Pyrrhocoris apterus* and *Dysdercus cingulatus* subjected to allatectomy and intraspecific or interspecific reimplantations of corpus allatum

Donor	Recipient	n	Oviposited females (%)	Pre-oviposited period (days)	Oviposited period (days)	Post-oviposited period (days)	Egg batches/ female	Eggs/ batch
P. apterus	*P. apterus*	8	87.5	7.0 (7)	21.7 (14–24)	2.3 (0–10)	6.4 (4–8)	53.9 (16–70)
P. apterus	*P. apterus* + P II	11	81.8	8.7 (6–13)	18.7 (0–28)	6.7 (0–23)	5.0 (1–7)	50.4 (22–70)
P. apterus	*D. cingulatus*	13	84.6	6.2 (5–11)	5.2 (0–13)	21.6 (7–30)	2.0 (1–4)	60.1 (13–90)
P. apterus	*D. cingulatus* + P II	10	60.0	7.5 (6–11)	8.2 (0–28)	18.8 (0–30)	1.3 (1–2)	22.6 (8–45)
D. cingulatus	*D. cingulatus*	7	85.7	6.3 (5–7)	18.2 (0–29)	4.5 (0–10)	5.0 (1–7)	47.4 (26–75)
D. cingulatus	*D. cingulatus* + P II	12	8.3	(24)	(0)	(11)	(1)	(28)
D. cingulatus	*P. apterus*	14	100.0	6.5 (5–7)	23.2 (9–30)	4.4 (0–13)	6.2 (3–8)	50.9 (6–74)
D. cingulatus	*P. apterus* + P II	11	54.6	9.0 (6–13)	23.3 (4–31)	4.0 (0–18)	6.0 (2–7)	46.5 (3–79)

Dose of P II per Petri dish: 600 µg. Experiments lasted 35 days.

reimplanted with *D. cingulatus* CA, the reproduction was inhibited in almost all females when the recipient was *D. cingulatus* and in less than 50% of females when the recipient was *P. apterus*.

Pratt et al[10] suggest that the enzymic competence of CA to oxidise precocene-like molecules to highly reactive epoxides is a basis of their selective cytotoxic action. The lack of such a competence might explain the low sensitivity of *P. apterus* CA to P II. Indeed, the metabolism of *P. apterus* CA seems to be different from other species; none of the classically known juvenile hormones has been found in reproducing females in a measurable concentration (Baehr, personal communication).

The physiological basis of the 'anti-precocene mechanisms' outside the CA can only be guessed at. There is evidence that P II penetrates to the body of *P. apterus* and can reach a concentration high enough to kill the animal. Thus the doses of 800 µg or 1000 µg of P II killed 40.0% (n = 10) or 73.3% (n = 18) of *P. apterus* females respectively within 1 week, although the surviving females still oviposited. In the species sensitive to P II, the concentration below the toxic level is sufficient to inhibit the CA[13]. Such a concentration should be achieved in the body of both *P. apterus* and *D. cingulatus* when the dose of 600 µg of P II (causing mortality in about 10% of individuals of both species) was used in the present experiments (tables 1 and 2, figure).

In *D. cingulatus* females P II suppresses the food intake in addition to its inhibitory effect on CA function[14]. Much smaller egg batches (by about 60%) oviposited by P II-treated *D. cingulatus* reimplanted by *P. apterus* CA (table 2) also indicate suppression of food intake or food utilization. In *P. apterus*, on the other hand, the treatment with P II did not markedly reduce the size of egg batches (tables 1 and 2). Even those *P. apterus* females surviving doses of P II as high as 800 µg or 1000 µg oviposited 47.5 (18-62) or 41.0 (36-45) eggs in the 1st batch respectively - a quantity which is not much lower than the normal egg batches in untreated females (tables 1 and 2). It appears, therefore, that *D. cingulatus* is more sensitive to the antifeedant effect of P II than *P. apterus*. Sláma[15] suggests that the antifeedant property of precocenes is a cause of their interference with the activation of endocrine glands. The inhibition of the implanted CA by P II, however, cannot be attributed only to its antifeedant effect as even completely starved *D. cingulatus* females with the denervated CA oviposited[16]. In contrast, the suppression of feeding by P II might favor its inhibitory effect on the CA. Conversely, a relatively high food intake in P II-treated *P. apterus* might decrease the inhibitory effect of P II on the CA. The CA function is generally stimulated by food intake, either directly or through the neurosecretory cells of the brain[17,18]. Such a stimulation might compete with the inhibition by P II. Although the physiological processes preventing the inhibition of ovarian development by P II in *P. apterus* cannot be precisely described so far, it has been proved that they are not limited to the CA itself.

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Effect of insulin-like growth factor on collagen and glycosaminoglycan synthesis by rabbit articular chondrocytes in culture

H.L. Guenther, Hannelore E. Guenther, E.R. Froesch and H. Fleisch[1]

Department of Pathophysiology, University of Berne, Murtenstrasse 35, CH-3010 Berne (Switzerland), and Department of Medicine, Kantonsspital, CH-8006 Zurich (Switzerland), 1 October 1981

Summary. Rabbit articular chondrocytes cultured in the presence of insulin-like growth factor I (IGF I) increased significantly the synthesis of both collagen and glycosaminoglycans. The increase in the ratio of $^{35}SO_4^{2-}$ to [^{3}H]glucosamine observed in glycosaminoglycans synthesized in the presence of IGF I seems to indicate that IGF I affects sulphation and synthesis of these polyanionic macromolecules to a different extent.

Insulin-like growth factor I (IGF I) a growth hormone dependent single-chain peptide was recently isolated from human plasma[2-5]. Its growth promoting activity on various tissues and cell systems[6-10], and its mol.wt of 7500 daltons, have assigned IGF I structurally and functionally to the class of the somatomedins[11]. In addition to its growth promoting abilities, IGF I was found to stimulate strongly the incorporation of sulphate into glycosaminoglycans of cartilage from various animal species[12,13]. Recently, it was shown that the synthesis of bone collagen was likewise increased by IGF I[14].

The purpose of the present study was to examine whether IGF I affects the rate of collagen synthesis by rabbit articular chondrocytes. Furthermore, based on previous findings[15] that brachymorphic mice, which have a mutation resulting in disproportionate dwarfism, preferentially synthesize low sulphated glycosaminoglycans, experiments were carried out to determine whether glycosaminoglycans synthesized by articular chondrocytes in the absence or presence of IGF I differ in their sulphate incorporation.

Materials and methods. Chondrocytes were prepared from the proximal femoral surfaces of 3 months old KUN FI