μCi of L-4,5-^{3}H-leucine (spec. act. 5 Ci/mmol) under a 95% oxygen: 5% carbon dioxide atmosphere. ^{3}H-leucine incorporation into proteins was estimated by measuring the radioactivity present in the trichloroacetic acid-insoluble residues[4]. Proteins concentration was measured according to LOWRY et al.[5].

Results and discussion. The administration of a single dose of estradiol caused a significant increase 24 h later of ^{3}H-leucine incorporation into neurohypophyseal proteins (Table). The effects of estradiol depended largely upon time of injection. Rats injected at 06.00 h, i.e., at the end of the dark period, exhibited a 74% increase in protein synthesis, whereas rats injected at 14.00 h, i.e. at the middle of the light period, showed only a 30% increase in protein synthesis.

Limited information is available concerning the possibility that estradiol treatment modifies the function of the neurohypophysis. In female rats, estrogens increase oxytocin content of the posterior lobe and gonadectomy reduces it slightly[6,7]. Data reported herein reveal that neurohypophyseal protein synthesis increases significantly after estradiol treatment. In addition, the time of day when estradiol was injected has a remarkable influence on the extent of stimulation of labelled amino acid incorporation into proteins. This may be related to the previously reported diurnal variations in estradiol uptake by the posterior lobe[3].

Time-dependence of the effects of estradiol on protein synthesis of the rat neurohypophysis

Treatment[a]	^{3}H-leucine incorporation into proteins (dpm/mg of protein)	
	06.00 h	14.00 h
Vehicle	612.0 ± 34.2[b]	591.1 ± 51.6
Estradiol	1065.3 ± 60.7[c]	769.8 ± 32.8[d]
Increase (%)	74.0	30.2

[a] Rats received a single injection of 0.3 ug of estradiol or vehicle at 06.00 or 14.00 h and were killed 24 h later. [b] Mean ± SE $n = 8$ in each group. [c] $p < 0.01$, Student's t-test. [d] $p < 0.05$.

Effects of estrogens on the neurons of the paraventricular nuclei are supported by neurophysiological[8] and autoradiographic[9] data. To what exent changes in oxytocin output reflects an effect of estradiol on hypothalamic or neurohypophyseal sites remains to be established. However, the present results, together with previous data[3], seem to argue in favour of the view that estrogens could affect the release of oxytocin in part by acting directly on the neurohypophysis.

Summary. The incorporation of ^{3}H-leucine into neurohypophyseal proteins was measured in vitro, 24 h after the administration of a single dose of estradiol (0.3 μg) to castrated female rats. Estradiol treatment caused a significant increase of ^{3}H-leucine incorporation into proteins of the posterior lobe. The effects of estradiol depended largely upon time injection. Rats injected at 06.00 h, i.e., at the end of the dark period exhibited a 74% increase in protein synthesis, whereas rat injected at 14.00 h, i.e., at the middle of the light period only showed a 30% of increase.

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Endocrine Control of Mating Instinct in *Dysdercus koenigii* (Hemiptera: Pyrrhocoridae)

In course of a study to investigate the endocrine pathway of the events occurring in the reproductive cycle of *Dysdercus koenigii*, a hemimetabolous insect pest in India on the common ladies finger plant (*Hibiscus esculentus*), a very interesting and remarkable relationship was observed between the neurosecretory cells of the brain and the urge for mating in the female.

Six paired groups of neurosecretory cells (NSC) occur in the protocerebrum of the brain of *Dysdercus koenigii*. Apart from the median neurosecretory cells (MNSC) forming 3 pairs of dorsal and 2 pairs of ventral groups, there is a lateral group of 3 neurosecretory cells (LNSC) on either side. The females mate within 24 h after emergence, may continue the act for 110 to 114 h and lay eggs about 120 h after commencement of mating. The receptivity of the female appears to be 'recognized' by the male which moves in the vicinity of the former and exhibits a courting behaviour. It has also been observed that mating must continue for at least 96 h for normal oocyte development and oviposition. During the first 96 h, the mating individuals are inseparably united and any interference during this period results in the failure of the females to lay eggs. Interruption of mating 96 h after commencement does not affect maturation and normal ovulation of eggs. Histological examination of paraldehyde – fuchsin stained sections passing through the protocerebrum to show the secretory activity of the brain NSC in various phases of the reproductive cycle has revealed that the LNSC are full of secretory material prior to, and at the time of commencement of mating, but after 24 h they are seen to be empty showing only a compact layer of secretory material at the periphery of the cells. There is no secretory activity in the MNSC of pars intercerebralis upto 72 h after commencement of mating. The activity of the LNSC completely subsides after mating and these cells have not been seen to enter a secretory phase even 10 days after oviposition. The insects probably never mate again after laying the first batch of eggs (no mating occurred in the females kept under observation for over a month, although they were quite active and feeding voraciously on the leaves of *Hibiscus*

esculentus). The appearance of LNSC in the 5th nymphal instar, their peak activity in the freshly emerged adult female prior to mating and their complete inactivity after mating and oviposition is highly suggestive of the fact that the LNSC secretion induces the urge for mating (receptivity) in the female. Males are attracted towards the female when the latter exhibits her mating instinct. Therefore, the sex attractant, if any, is produced by the female and the effect of this attractant is to engage the male in copula for at least 96 h. There is sufficient evidence[1] to show that there is a definite relationship between mating and the activity of the corpora allata (CA).

Different views have been expressed regarding the control of mating in insects. ENGELMAN[2] and BARTH[3–5] have stated that mating is controlled by CA. ROTH[6] and ROTH and BARTH[7] showed that the female receptivity was not controlled by the CA or the ovaries and came to the conclusion that a receptivity centre of the brain NSC controlled the acceptance of male by a female and that the act of copulation rendered the receptivity centre inactive. It is, therefore, logical to regard the LNSC of protocerebrum of female *D. koenigii* as the receptivity centre, the endocrine secretion of which induces the onset of receptivity in the freshly emerged female.

Summary. Five pairs of median and 1 pair of lateral neurosecretory cell groups occur in the protocerebrum of *Dysdercus koenigii*, a hemipteran pest on the ladies finger plant (*Hibiscus esculentus*). The lateral neurosecretory cells (LNSC) become active prior to, and at the time of commencement of mating and release their secretion within 24 h of commencement. The female never mates again after laying eggs and the LNSC also never become active. It is believed that LNSC secretion induces the urge for mating in the freshly emerged female and the lateral groups of NSC form the receptivity centre in the brain.

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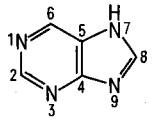
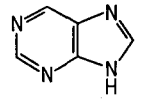
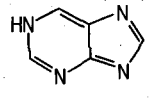
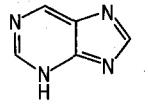
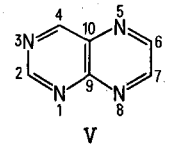
THEORIA

On the Use of π-Excessive and π-Deficient Terminology for Heterocyclic Bases

ALBERT[1] has proposed a useful classification of nitrogen heterocycles into π-excessive and π-deficient systems. Briefly, 5-membered rings bearing an –NH– group, e.g. pyrrole, pyrazole and imidazole, belong to the first type (π-excessive) while 6-membered rings such as pyridine, pyrimidine and pyrazine belong to the second type. However, from these definitions it is not at all clear how these monocycles should be classified when fused to form a new hetero system. For example, can the pyrimidine moiety of purine-NH-1 or NH-3 tautomeric form still be regarded as a π-deficient ring? Conversely, which of the 2 ring systems embodied in pteridine is 'more' π-deficient (pyrimidine or pyrazine)?

Compound	Fragment	Total π charge (CNDO/2)
 I	C_6-N_1-C_2-N_3	−0.139
	N_7-C_8-N_9	+0.181
	C_4-C_5	−0.042
 II	C_6-N_1-C_2-N_3	−0.137
	N_7-C_8-N_9	+0.177
	C_4-C_5	−0.040
 III	C_6-N_1-C_2-N_3	+0.289
	N_7-C_8-N_9	−0.368
	C_4-C_5	+0.079
 IV	C_6-N_1-C_2-N_3	+0.346
	N_7-C_8-N_9	−0.420
	C_4-C_5	+0.074
 V	N_1-C_2-N_3-C_4	−0.018
	N_5-C_6-C_7-N_8	+0.001
	C_9-C_{10}	+0.017

The CNDO/2 method, known to give reliable charge distributions[2,3], provided the following data (Table):

a) Purine tautomers I, II, bearing an imidazole ring in its π-excessive state according to ALBERT's proposal, possess a *positive* π charge on the fragment N_7-C_8N_9 and a *negative* π charge on the pyrimidine counterpart C_6-N_1-C_2-N_3. The bridged carbon atoms C_4-C_5, common to both rings, serve as a negatively-charged sentinel.

b) The reverse is observed for purine tautomers III, IV in which the imidazole is in a quinoid-like structure. Here the N_7-C_8-N_9 fragment is *π-negative*, while the pyrimidine analogue C_6-N_1-C_2-N_3 is *π-positive*. However, C_4-C_5 accomodates a positive character.

c) In pteridine (V), the pyrimidine framework N_1-C_2-N_3-C_4 is π *negatively* charged, while the pyrazine counterpart N_5-C_6-C_7-N_8 is slightly positive. The negative π charge on the pyrimidine fragment is derived mainly from the bridged C_9-C_{10} atoms.

From these and related CNDO/2 calculations[4], the following conclusions may be deduced: 1. Where no

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