shown that a dose of 0.1 µmol intraventricular GABA inhibited Prl release whereas a 4-µmol dose elevated Prl levels[6,8,12]. Castration reduced GABA levels in different brain regions and AOAA produced only a moderate increase of GABA in these rats[13]. The inhibition of Prl release by AOAA could possibly be due to the failure of this compound at the dose used here to elevate endogenous brain GABA levels to the extent of 4 µmol to produce an increase in Prl release. On the other hand, this dose of AOAA has induced an increase in the level of GABA that is capable of inhibiting Prl release. Intravenous infusion of AOAA failed to affect Prl levels in castrated male rats, but inhibited a sulpiride-induced rise in Prl[14].

The increased TH activity indicates that GABA concentrations induced by AOAA at this dose can stimulate de novo synthesis of hypothalamic dopamine (DA), which can be released from tubero-infundibular dopaminergic nerve terminals located in the median eminence into the hypophyseal portal blood and directly inhibit AP Prl release. This is supported by the fact that DA levels in hypophyseal portal blood are sufficiently high to have a direct action on pituitary to inhibit Prl release[15]. Aminooxyacetic acid is also shown to increase DA levels in nigro-striatal dopaminergic neurons[16]. A direct action of GABA on Prl release is possible since GABA receptors are located in the pituitary gland[17]. Aminooxyacetic acid failed to affect LH and FSH levels, and only very high doses of GABA resulted in stimulation of LH release[8]. The present observation supports the hypothesis that GABA is involved in the regulation of Prl release and appears to act through hypothalamic dopamine.

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Neuroendocrine control of carbohydrate metabolism in the freshwater bivalve mollusc *Lamellidens marginalis*

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Summary. Blood sugars and foot muscle glycogen were measured in the mussel, *L. marginalis* after ablation of the cerebral ganglia, and in mussels injected with cerebral ganglionic extract 3 h after ablation. There is a rise in the blood sugar and decrease in foot muscle glycogen 3 h after operation, but no change in sham-operated controls. The effect of ablation is reversed by injecting brain extract into ablated mussels. No such effect could be seen in the controls. The results are suggestive of the presence, in the cerebral ganglia, of a hypoglycaemic factor similar to insulin.
Key words. Mussel; *Lamellidens marginalis;* carbohydrate metabolism, neuro-endocrine control of; foot muscle glycogen; blood sugar; glycogen, foot muscle; hypoglycemic factor.

The neuroendocrine regulation of metabolism in pelecypod molluscs has received scant attention. The presence of neurosecretory cells in the central ganglia of a few bivalves has been reported[1-3], but their involvement in metabolic regulation is poorly understood. A cardioexcitatory neuropeptide has been isolated from the central ganglia of a bivalve[4], and the gut epithelium and hepatopancreas of some bivalves have been implicated in the production of insulin-like substances and control of carbohydrate metabolism[5,6]. However, nothing is known about metabolic regulation by the central ganglia of bivalves.

The present work is a report on the role of the cerebral ganglia of *L. marginalis* in regulating carbohydrate levels in the hemolymph and foot muscle.

Materials and methods. The mussels, *L. marginalis* of uniform size collected from ponds in Anantapur district were maintained in aquaria. After careful removal of one of the shell valves, the mussels were maintained for one week prior to experimentation. Ciliary activity of ctenidia and rate of heart beat were taken as indices of the health of the mussels after shell valve removal. The ablation of cerebral ganglia was done according to the method given by Nagabhushanam[7]. Control mussels were sham-operated in such a way that cerebral ganglia remained intact. A buffer extract (phosphate buffer pH 7.4) of cerebral ganglia from 15 mussels was prepared in a final volume of 3.0 ml. 0.1 ml of the extract was injected into the foot muscle of each mussel 3 h after ablation. Two sets of ablated mussels received separately 0.1 ml of phosphate buffer (pH 7.4) and 0.1 ml foot muscle extract prepared in phosphate buffer (pH 7.4), and served as controls. The sham-operated mussels were grouped into three sets and received separately brain extract, phosphate buffer (pH 7.4) and foot muscle extract.

Hemolymph was collected from the heart 1 h, 2 h, and 3 h after ablation and 30 min and 1 h after injection, as well as from normal unoperated mussels, centrifuged at 3000 rpm for 10 min to remove cellular elements, and total sugars were determined as total anthrone positive substances[8]. Foot muscle was isolated from each mussel and dried in a hot-air oven at 80 °C to constant weight, and glycogen was determined by the anthrone method[9]. The data were subject to statistical validation using Student's t-test for significance[10].

Results. There was a rise in blood sugar and decrease in foot muscle glycogen during 3 h following ablation of cerebral gan-

glia. These effects were reversed by injection of the brain extract (table). Injection of brain extract into sham-operated mussels resulted in lowering of blood sugars and a rise in foot muscle glycogen.

Discussion. The results indicate the possible presence, in the cerebral ganglia of the mussel *L. marginalis*, of a neurohormonal substance that has a regulatory effect on carbohydrate metabolism. The changes in carbohydrate levels induced by ablation and subsequent injection of the extract into ablated and sham-operated mussels suggest that the factor is hypoglycemic, having an activity similar to vertebrate insulin.

Effect of ablation of cerebral ganglia and injection of cerebral ganglionic extract on the carbohydrate levels in the hemolymph and foot muscle of the mussel *L. marginalis*. The values are mean ± SD for 10 animals. p-Values significant, * < 0.01

	Time	Total blood sugar mg/100 ml Mean ± SD	Foot glycogen g/100 g dry tissue Mean ± SD
Normal mussels	–	40.1 ± 8.7	11.0 ± 0.2
Sham operated controls	1h	41.2 ± 4.2	11.2 ± 0.5
	2h	42.1 ± 5.8	11.8 ± 0.6
	3h	41.0 ± 6.5	11.2 ± 0.8
Ablated mussels	1h	57.0 ± 5.6*	10.3 ± 2.8*
	2h	58.0 ± 4.2*	9.5 ± 4.3*
	3h	58.2 ± 2.0*	9.1 ± 4.1*
Ablated mussels injected with phosphate buffer pH 7.4 (control)	30 min	59.0 ± 3.4	8.8 ± 2.8
	1h	58.9 ± 4.9	9.2 ± 2.7
Ablated mussels injected with foot muscle extract (control)	30 min	60.2 ± 2.9	8.7 ± 3.2
	1h	59.5 ± 3.8	8.9 + 3.5
Ablated mussels injected with brain extract	30 min	47.0 ± 0.7*	9.3 ± 2.8*
	1h	38.6 ± 9.2	10.7 ± 2.8
Sham operated mussels injected with brain extract	30 min	35.4 ± 7.5*	12.8 ± 1.2*
	1h	33.8 ± 6.5*	13.4 ± 0.8*
Sham operated mussels injected with phosphate buffer (pH 7.4) (control)	30 min	41.8 ± 6.5	11.0 ± 1.1
	1h	42.1 ± 5.6	10.7 ± 0.9
Sham operated mussels injected with foot muscle extract (control)	30 min	40.5 ± 3.8	10.8 ± 2.2
	1h	41.5 ± 7.2	11.5 ± 3.8

The physiological role of neurosecretion in pelecypod molluscs is poorly understood, although there is some evidence for the presence of neurosecretory cells in the central ganglia of a few pelecypods[1-3]. Since several gastropod and pelecypod molluscs have been found to have carbohydrate-oriented metabolism[11-13], molluscan tissues should be able to synthesize and store carbohydrate as glycogen, and utilize it under stress. This is possible, perhaps under the aegis of hormonal substances. Vertebrate insulin was found to cause hypoglycemia and glycogen synthesis in the tissues of the clam *Meretrix costa*[13], indicating that molluscan tissue is responsive to insulin. In a search for the source of a hypoglycemic factor in molluscan tissues, the gut wall of pelecypods *Anodonta cygnea, Unio pictorum* and *Ostrea edulis* was found to produce a factor similar in activity to that of vertebrate insulin[5,6,15,16]. The present work has revealed yet another source for a hypoglycemic factor. This opens up possibilities for regulation of carbohydrate metabolism at different levels by multiple hormones. However, a generalization is not possible at this stage as further probing into other sources in molluscan tissues is needed.

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Nuclear progestin receptor in the chimpanzee sex skin[1]

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Summary. We describe for the first time the presence of a nuclear progestin receptor in the sex skin of the chimpanzee, which is possibly involved in the anti-estrogen action of progestins in the sexual swelling.
Key words. Chimpanzee; sex skin; progestin receptor; anti-estrogen action.

The sex skin of the chimpanzee undergoes a cyclic fluctuation in its degree of swelling[3]. The degree of sexual swelling has been correlated with the concentration of the nuclear estrogen receptor in the tissue, which characterizes the chimpanzee sex skin as an estrogen target tissue[4]. Since progestins inhibit estrogen-induced swelling, an anti-estrogen action has been suggested for progestins in the sexual swelling of the chimpanzee[4].

As a preliminary step to determine whether or not this anti-estrogen action of progestins is a receptor-mediated action of the steroid, we investigated the presence of the nuclear progestin receptor in the chimpanzee sex skin.

Materials and methods. Sex skin tissues (0.7–1.8 g) biopsied from chimpanzees at various reproductive stages were homogenized at 0–4 °C in 4 vol. of TEDG (10 mM Tris-HCl, 1 mM EDTA, 12 mM monothioglycerol, 10% (v/v)