totally inhibited the uptake of ^{125}I-hFSH indicating specificity of ^{125}I-hFSH uptake by immature rat testis (table).

Discussion. Several reports have appeared on the possible mode of action of inhibin at the level of pituitary[4,6,13,14] and/or hypothalamus[7,14,15]. The results of the present in vivo studies demonstrate that inhibin could significantly affect the FSH uptake by immature rat testicular tissue. The data confirm our earlier report that under in vitro conditions human seminal plasma inhibin suppresses the binding of FSH to testis[19].

It has also been shown that inhibin, both of ovarian and testicular origin, suppresses the binding of FSH to testis and the accumulation of cyclic AMP by testis[7], which suggests that inhibin modulates FSH action at the gonadal level. Irrespective of the mol.wt of inhibin (ram: 1500 daltons, human: 19,000 daltons) a similar action was observed indicating presence of a common biologically active moiety in both preparations.

The present findings suggest that inhibin has multiple sites of action and further knowledge on these lines may lead to our understanding of various reproductive dysfunctions.

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Insulin stimulates sodium-potassium activated ATPase from rat hippocampus[1]

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Summary. It is suggested that insulin is capable of activating cerebral NaK-ATPase in a dose-dependent manner.

There is growing interest in a possible physiological relevance of insulin for the central nervous system since it has become evident that nervous tissue contains not only insulin-specific receptors[2-5] but also the hormone itself[6-8]. From findings that insulin does not facilitate the transport of glucose through the blood-brain barrier[9] (BBB), it was concluded that insulin probably fails to act on any kind of brain function. However, the presence of insulin-binding sites in blood vessels[5] and the demonstrated ability of the hormone to stimulate potassium uptake by the brain[10], as well as data about a direct action of insulin on plasma membrane NaK-ATPase in lymphocytes[11], encouraged us to study the effect of the hormone on cerebral NaK-ATPase.

Material and methods. Hippocampi were isolated from rat brain, homogenized in 50 mM Tris HCl buffer (pH 7.4) and centrifuged (20000 × g, 60 min). For comparison, samples were taken from hypothalamus and treated analogously. The determination of ATPase activity was carried out in resuspended sediments from the procedure described above, using the method of Adam-Vizi et al.[12]. Estimation of P_i liberation was performed by the method of Eibl and Lands[13], slightly modified by the authors. Insulin was tested in the following concentrations: 60 μU/ml, 80 μU/ml, 100 μU/ml, 125 μU/ml, 500 μU/ml and, as a really pharmacological reference dose, 20,000 μU/ml. To ensure the effects of insulin on enzymatic activity, an anti-insulin serum with appropriate properties (generated in guinea-pigs) was used as a control substance to paralyze insulin action. The antiserum was utilized diluted 1:100 in distilled water. The protein content of the samples was estimated by the method of Lowry[14]. The statistical treatment of the data was performed by the non-parametric U-test.

Results. The influence of insulin on the activity of sodium-potassium ATPase is summarized in the table. Insulin acts in a dose-dependent manner. At 60 μU/ml the activity was significantly lower than in the controls. 80 μU/ml 'norma-

The effect of various concentrations of insulin on cerebral ATPase

	Insulin (μU/ml)	Percent of basal activity	Significance
Hippocampus	Basal activity: 1.143 ± 0.078 m kat/kg		
	Without	100	-
	60	78	$p < 0.02$
	80	103	no
	100	122	$p < 0.02$
	125	142	$p < 0.01$
	500	172	$p < 0.001$
Hypothalamus	Basal activity: 1.056 ± 0.107 m kat/kg		
	100	129	$p < 0.02$

lized' ATPase activity. At concentrations of 100 μU/ml and higher an enhancement of the ATP hydrolysis could be observed. The highest increase was at 500 μU/ml (172% of the normal activity). 20,000 μU/ml did not affect the enzyme-caused P_i liberation (not shown in the table). The behavior of hypothalamus-derived ATPase was identical. The addition of antiinsulin serum reduced the activity of ATPase to the control level, thus clearly indicating that the effects registered are due to the presence of the hormone.

Discussion. The demonstrated effect of insulin on cerebral NaK-ATPase is in contrast to reports which have shown the failure of insulin to influence ion fluxes in the brain[15,16]. The observed similar behavior of ATPase from hippocampus (a CNS region with an expressed blood-brain barrier) and hypothalamus (lacking a BBB) indicates that the stimulatory influence of insulin is not restricted to the blood vessel-associated part of ATPase activity, which is possibly involved in the maintenance of BBB. There are a few reports dealing with insulinbinding sites at synaptosomes derived from rat CNS[17,18] and axonal terminals[19]. Since nerve endings and the axolemma are densely populated with NaK-ATPase molecules it cannot be excluded that the effect of the hormone was directed to these neuronal particles. Recently, an effect of insulin on the activity of another enzyme in the CNS, ornithine decarboxylase, was suggested[20]. In any case the functional consequences of these findings will remain unclear until new data appear that can help to clarify the significance of insulin for brain metabolism.

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Radiochemical assays of corpus allatum activity in adult female cockroaches following ovariectomy in the last nymphal instar

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Summary. Surgical removal of the ovaries from nymphal *Periplaneta americana* results in lower than normal corpus allatum activity in the adult insect and an apparent absence of the manifestly cyclic pattern of juvenile hormone biosynthesis found in intact mated females. The results suggest that the presence of synchronously developing ovaries is necessary for the attainment of normal synthetic activity in the corpus allatum of this species.

It has been demonstrated that juvenile hormone (JH) is necessary for both the synthesis of vitellogenins and their uptake into the oocytes in *Periplaneta americana,* and radiochemical determinations of JH biosynthesis by isolated corpora allata (CA) indicate that the glands of adult females undergo cycles of activity in vivo[3,4]. The peaks of JH biosynthesis correspond temporally with the later stages of vitellogenesis of terminal oocytes and, at the same time, with the early stages of vitellogenesis of penultimate oocytes[3,4], but the precise physiological basis of the cycles of hormone production is imperfectly understood and the regulatory factors are unknown. Previous studies have proposed feedback loops between the ovaries and the CA in a variety of insect species[5-7] but, with a single exception[7], these have been limited to measurements of glandular size or to histological appearance which are not necessarily indicative of precise glandular activity[8]. In this paper we report the effects of ovariectomy on JH biosynthesis by the CA from *P. americana* which is part of a programme to fully investigate the control of reproduction in this species.

Materials and methods. Last (5th) nymphal instar *P. americana* were anaesthetized with nitrogen, surface sterilized with ethanol, and surgically ovariectomized through 2 small incisions in the dorsal cuticle of abdominal segment VII. The wounds were then sealed with wax. Antibiotics were not used, but survival and successful moulting of operated insects was better than 80%. The operations were performed in the nymphal stage in order to allow time for any artefactual disturbance to dissipate. The stimulatory effects of sham surgery on cockroaches appear to be short-lived[7] and it was therefore considered unnecessary to perform such operations on controls. When moulting to the adult instar had occurred the operated insects were maintained with intact male and female adults under conditions previously described[3,4].

Ovariectomized and control adult females were sacrificed at various times after adult emergence and activities of their CA assessed by the incorporation of radiolabel from methyl ^{14}C-methionine (Radiochemical Centre, Amersham, U.K.) into JH III in vitro using published methods[3,4].