Table 2 shows average pup weights at day 5-6 of lactation. Treating lactating rats with CB-154 significantly inhibited milk secretion as judged by the failure of the pups to increase b.wt ($p < 0.001$).

Discussion. Although the absorptive function of the small intestine has been studied in depth, the factors which regulate gut hypertrophy during certain states, such as lactation, have received less attention. Several reports have appeared on increases in gut weights during lactation[4,5,8-10]. In addition to increased gut weight, increases in gut nitrogen content during lactation have also been reported[9].

The present observations have shown that pregnancy exerts only a limited effect on intestinal weight, whereas lactation causes greater increases in intestinal weights. It has been suggested that increases in gut weight are dependent on the duration of lactation and on the number of suckling pups[8]. Furthermore, parturition which is unaccompanied by suckling has no effect on gut weight[9].

A definite stimulus for prolactin secretion during p.p. lactation is suckling[12-14]. These authors showed that after parturition plasma prolactin levels rose to peak values on day 5 p.p. and then gradually decreased to low levels at weaning. They also showed that during pregnancy plasma prolactin levels was elevated only for the first 3 days and then declined to low levels until the last day of pregnancy. Whereas CB-154 had no effect on intestinal weights during mid-pregnancy, it caused dramatic reductions in intestinal weights during lactation suggesting the involvement of prolactin.

2-bromo-α-ergrocryptine is an important inhibitor of prolactin secretion[15-17] and has been shown drastically to inhibit lactation when appropriate doses are administered to lactating animals[18]. Inability of the CB-154-treated lactating rats to raise young, judged by the failure of the litter to gain weight, strongly suggests that prolactin secretion was inhibited by CB-154.

It is suggested that changes in gut weights associated with pregnancy and especially lactation can be explained on the basis of increased prolactin secretion. Furthermore, it has been claimed that prolactin enhances food intake much more in females than in males[19]. Prolactin secretion might therefore explain the increased gut weight seen during lactation.

Table 1. Effect of pregnancy, lactation and 2-bromo-α-ergocryptine on jejunal wet weights (means ± SEM)

Jejunal segment	Physiological state and treatment	Number of rats	Wet weight (g)
III	Dioestrus	15	0.73 ± 0.02
	Pregnant	17	0.77 ± 0.01
	Pregnant + CB-154	13	0.75 ± 0.02
	Lactating	13	0.94 ± 0.04[c]
	Lactating + CB-154	12	0.80 ± 0.03[a]
IV	Dioestrus	15	0.72 ± 0.02
	Pregnant	17	0.77 ± 0.02[a]
	Pregnant + CB-154	13	0.78 ± 0.02[a]
	Lactating	13	0.97 ± 0.03[c]
	Lactating + CB-154	12	0.83 ± 0.02[b,c]

Significance of differences as compared with dioestrus rats: [a] $p < 0.05$, [b] $p < 0.01$ as compared with lactating rats, [c] $p < 0.001$.

Table 2. Effect of 2-bromo-α-ergocryptine administration to lactating rats on the body weights of pups (means ± SEM)

Group and treatment	Number of rats	Body weight of pups (g)	
Lactating	7	13.86 ± 0.46	$p < 0.001$
Lactating + CB-154	8	7.88 ± 0.45	

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4 I.L. Craft, Clin. Sci. *38*, 287 (1970).
5 A.W. Cripps and V.J. Williams, Br. J. Nutr. *33*, 17 (1975).
6 J.R. Mainoya, J. Endocr. *67*, 351 (1975).
7 B.A. Rolls, Br. J. Nutr. *33*, 1 (1975).
8 A.B. Cairnie and R.E. Bentley, Exp. Cell Res. *46*, 428 (1967).
9 B.F. Fell, K.A. Smith and R.M. Campell, J. Path. Bact. *85*, 179 (1963).
10 C.P. Sigdestad and J.W. Osborne, Growth *36*, 165 (1972).
11 D.F. Horrobin, Prolactin, p. 208. Eden Press, Montreal 1976.
12 Y. Amenomori, C.L. Chen and J. Meites, Endocrinology *86*, 506 (1970).
13 J. Meites, H.K. Lu, W. Wuttke, C.W. Welsch, H. Nagasawa and S.K. Quadri, Recent Prog. Horm. Res. *28*, 471 (1972).
14 A.A. Simpson, M.H.W. Simpson, Y.N. Sinha and H.G. Schmidt, J. Endocr. *58*, 675 (1973).
15 E. Flückiger and E. Kovacs, Experientia *30*, 1173 (1974).
16 R. Yanai and H. Nagasawa, Horm. Res. *5*, 1 (1974).
17 J.A. Clemens, C.J. Shaar, B. Smalstig, N.J. Bach and E.C. Kornfeld, Endocrinology *94*, 1171 (1974).
18 C.J. Shaar and J.A. Clemens, Endocrinology *90*, 285 (1972).
19 G.A. Bray, Fedn Proc. *33*, 1140 (1974).

The effect of glycine on serum luteinizing hormone in adult female rats[1]

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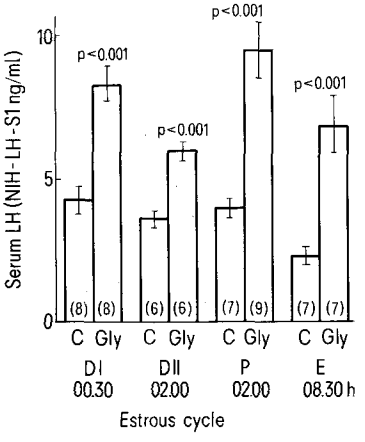
Summary. The response of serum luteinizing hormone to glycine has been studied during the estrous cycle in adult female rats. I.p. administration of 200 mg of glycine significantly elevated serum luteinizing hormone levels at all stages of the estrous cycle.

Following the original proposal by Aprison and Werman[2] based on the studies of the regional distribution of glycine in cat spinal cord, much evidence indicating that glycine is an neurotransmitter in the mammalian central nervous system has been accumulating[3-6]. In the previous experiments, authors demonstrated that the concentration of glycine in only the middle hypothalamus (including the median eminence, arcuate and ventromedial nucleus) significantly increased during the critical period[7], and that the content of luteinizing hormone releasing hormone (LH-

RH) in the middle hypothalamus significantly decreased during the critical period[8]. The present experiment was performed to investigate the effect of glycine on serum luteinizing hormone (LH) in adult female rats.

Materials and methods. Experiments were carried out on adult female rats, weighing 250–300 g. Laboratory chow and tap water were provided ad libitum. The animals were kept under reversed lighting (light from 22.00 h–10.00 h) for 30 days before the starting to take vaginal smears. Under our laboratory conditions, the critical period is around 05.00 h–08.00 h on proestrus[9]. Daily vaginal smears were taken before 23.45 h until autopsy. Each of the rats selected had shown at least 3 consecutive 4-day estrous cycles before the cycle when it was used. Glycine was dissolved in 0.9% NaCl. 200 mg of glycine or 0.9% NaCl in a volume of 1 ml was injected i.p. into each of the 4-day cyclic rats at various times throughout the estrous cycle. 20 min after injection, the animals were exsanguinated by decapitation. After centrifugation the serum was kept frozen at −20 °C until the day of assay. LH was measured by radioimmunoassay using NIAMDD reagents. The samples were assayed in duplicate with an intra-assay coefficient of variation of less than 5%. All samples from each experiment were run in the same radioimmunoassay.



Serum LH levels 20 min after injection of 0.9% NaCl or glycine at various stages of the estrous cycle. Vertical lines indicate ±SEM, and the number of rats is given in parentheses. p-values are for control vs experimental in each experiment. Abbreviations are as follows: C = 0.9% NaCl, Gly = glycine, D_I = diestrus I, D_{II} = diestrus II, P = proestrus, E = estrus.

Results and discussion. The results are shown in the figure. At all stages of the estrous cycle, the i.p. injection of 200 mg of glycine produced significant increases in serum LH levels ($p < 0.001$). However, only the small changes in the response at different stages could be observed. The response was minimal on diestrus II and maximal on proestrus. The results of the present study demonstrate that the acute administration of glycine elevates serum LH levels in adult female rats. Tada et al.[10] observed that glycine injected i.p. passed the blood-brain barrier. The present results together, with the previous observations[7,8], suggest the possibility that the site of action of glycine may be within the middle hypothalamus and glycine may play a role in regulating the LH-RH release. However, Ondo et al.[11] demonstrated that serum LH levels did not increase following the intraventricular or intrapituitary administration of glycine. The discrepancy between our present results and the results of Ondo et al. may depend on the different uptake site of glycine following the different administration. Further experimentation is required to find the site in the central nervous system where glycine causes the increased LH release.

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2 M.H. Aprison and R. Werman, Life Sci. *4*, 2075 (1965).
3 D.R. Curtis, A.W. Duggan and G.A.R. Johnston, Brain Res. *14*, 759 (1969).
4 L.L. Iversen and F.E. Bloom, Brain Res. *41*, 131 (1972).
5 W.J. Logan and S.H. Snyder, Brain Res. *42*, 413 (1972).
6 J.-C. Reubi and M. Cuénod, Brain Res. *112*, 347 (1976).
7 H. Morishita, S. Kuroiwa, M. Tomioka, K. Higuchi, H. Mitani, N. Nagamachi, M. Kawamoto, T. Ozasa and H. Adachi, Brain Res. *104*, 363 (1976).
8 I. Oshima, H. Morishita, K. Omura and S. Saito, submitted for publication.
9 H. Morishita, N. Nagamachi, M. Kawamoto, J. Yoshida, T. Ozasa and H. Adachi, Acta endocr. *71*, 226 (1972).
10 K. Tada, G. Takada and T. Arakawa, Tohoku J. exp. Med. *103*, 49 (1971).
11 J.G. Ondo, K.A. Pass and R. Baldwin, Neuroendocrinology *21*, 79 (1976).

Evidence for in vitro release of neurophysin by the rat pineal gland

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Summary. Cultured pineal ependymal cells from rat fetuses release into their incubation media immunoreactive neurophysin. The presence of neurophysin was assessed by radioimmunoassay. The culture medium was found to contain 440 pg neurophysin per mg protein.

We have observed immunoreactive neurophysins in both bovine[1] and human[2] pineal glands. 2 immunoreactive neurophysins, distinguished by their electrophoretic mobility and their antigenic properties, were present in each species. The pineal neurophysins also share many characteristics with the pituitary neurophysins of the same species. The present communication reports the presence of immunoreactive neurophysin in the media of cultured pineal cells from rat fetuses.

Materials and methods. Lyophilized culture media from pineal cells of rat fetuses[3] were supplied by Dr Pavel. This method is detailed elsewhere[3] and is summarized below. The cells corresponding to each gland were suspended in 1 ml of Hank's medium supplemented with 10% calf serum and 2.5% N16 medium. On day 8, when the cell cultures were established, the media were first changed and thereafter they were changed every 5 days. Lyophilized media from 18-day-old fetal pineal cells representing 6.2 mg protein per ml were dissolved in 0.9% NaCl. The medium without cells served as control. The neurophysin was detected by radioimmunoassay (RIA) with an antibody directed against bovine neurophysin II (bNII). This antibody displays a low specificity in that it recognized not only bNII but also one of the neurophysins from man and rat.