($PB^{131}I\%$) in thyroidectomized chickens was as high as in intact controls, 72 and 96 h after a radioiodine injection.
However, in recent experiments (unpublished data) using gel Sephadex column fractionation, we could show that in thyroidectomized ducks: protein-bound radioactivity was still present in the plasma but this PBI included 100% of non-hormonal iodine. It therefore seems unquestionable that total thyroidectomy removes the only source of thyroid hormone in birds as in other species.
Conclusion. The competitive protein-binding procedure appeared to provide a reliable method for the study of avian thyroid function. The results presented here on drakes showed (a) low levels in plasma T4 compared with the rat, (b) striking seasonal changes in animals reared outdoors, (c) marked variations in animals exposed to environmental conditions that can be considered as rather common for wild life animals, e.g., temporary deprivation of food (i.e. during migration or egg incubation), and cold. Whereas PBI was unable to detect any modification in thyroid function, the T4 binding method showed significant alterations of thyroid activity in such circumstances. And finally, the method can be useful in testing the effectiveness of thyroidectomy, while PBI determinations are definitely inadequate in this respect.

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Possible influence of prolactin on intestinal hypertrophy in pregnant and lactating rats[1]

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Summary. The increase in intestinal weights during lactation, and to a lesser extent during pregnancy, is inhibited by bromocriptin. This suggests that increased prolactin secretion might be responsible for gut hypertrophy during lactation.

It is now evident that gestational and lactational states may modify intestinal absorption of electrolytes and nonelectrolytes[4-6]. Recently, it was reported that rat intestinal dipeptidase activity increased slightly during pregnancy and much more during lactation[7]. Available data indicate that the small intestine shows important increases in weight in lactation[4,5,8-10] which are not always observed during pregnancy[4,5]. Some observations suggest that during lactation intestinal weight and length progressively increase to a maximum a few days before weaning and decrease to normal values about 30 days post partum[5,9].
Although there is a definite relationship between gut weight changes and the period of lactation[5,9], little is known about the mechanisms which control gut hypertrophy during lactation. Increased food intake has been attributed to the observed gut hypertrophy and hyperplasia[9]. Recently, it has been suggested that the effects of pregnancy and lactation on gut functions, such as gut hypertrophy observed during lactation, could be mediated by prolactin[11]. The purpose of the present note is to report on the results of an investigation to find out whether the gut hypertrophy seen during lactation is attributable to increased prolactin secretion.
Materials and methods. In the present study, nonfasted 3-month-old Sprague Dawley rats (Simonsen Labs. Gilroy, California) were used. They were maintained on a 'white diet' (Simonsen Lab.), food and water given ad libitum.
The groups of rats used included: Dioestrous rats, rats at day 12-13 of pregnancy, mid-pregnant rats pretreated once daily for 5 days with 60 µg of 2-bromo-α-ergocryptine (= bromocriptin or CB-154, Sandoz), rats between day 5 and 6 of lactation nursing 8 pups, and rats nursing 8 pups pretreated once daily from day 1 of p.p. for 5 days with 60 µg of CB-154. Bromocriptin (60 µg) was suspended in physiological saline and injected s.c. to mid-pregnant and lactating rats.
Following nembutal anaesthesia, 2 12-cm long segments from the mid-jejunum (segments III and IV) were removed, rinsed out with physiological saline, quickly blotted on filter paper and weighed on a Microanalytical Mettler-H-balance. Each litter was weighed and average weight of pup determined per lactating rat. The weights are given in g and expressed as means ± SEM. All statistical comparisons were made with a t-test.
Results. As shown in table 1, jejunal weight increases during mid-pregnancy only reached significant levels in segment IV ($p < 0.05$). Pregnant rats pretreated for 5 days with CB-154 had jejunal weights which did not differ from dioestrous controls. However, 5 days lactation caused significant increases in both segments ($p < 0.001$) as compared with dioestrous rats. But lactating rats treated with CB-154 from day 1 p.p. for 5 days showed significant decreases in jejunal weights compared with untreated lactating rats ($p < 0.01$).

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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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-