There was significant reduction in the liver vitamin A stores in chronically alcoholic rats without any change in plasma vitamin A levels. These observations are in agreement with those of Blomstrand et al[4]. Treatment of alcoholic rats with PTU for 2 weeks restored the liver vitamin A levels to control values.

Many hormonal and environmental factors influence the physiological requirement, metabolism and storage of vitamin A, of which adrenal activity and thyroid status have been shown to be particularly important[10]. Both these endocrine glands are known to be stimulated by ethanol[11-14]. Clark and Colburn[15] have shown that in male rats on vitamin A-deficient diet, injections of cortisone drastically reduced the total liver reserves of vitamin A. Conversely vitamin A has been shown to be involved in corticosteroid biosynthesis from cholesterol in rat adrenals[16], thus increasing the physiological requirement and utilization. The effect of thyroxine in inducing vitamin A deficiency has been hinted[17,18]. In fact there appears to be an inverse relationship between vitamin A status and thyroid status in rat[19]. Recently studies in chicken have shown that the release of vitamin A into circulation is interfered with, in thyroxine-treated birds causing low plasma levels due to inadequate availability of retinol-binding protein. This has been shown to be the result of enhanced plasma turnover rate, uncompensated for by synthesis[20]. This, however, does not appear to be the case in rats, since the plasma vitamin A levels were not significantly different from controls. Alcohol has been implicated as a factor capable of mobilizing vitamin A into blood from livers of animals[1]. Thus, the activation of pituitary-thyroid and pituitary-adrenal axis may be a responsible factor for the reduction of liver vitamin A stores in chronically alcoholic rats.

This is the first report on the beneficial action of PTU in reversing the alcohol-induced reduction of liver vitamin A. Earlier we had found a similar action on plasma protein synthesis (to be published). The mode of action of PTU is not very clear at present. It may be due to a direct antithyroid effect, coupled with indirect extra-thyroidal action.

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Effect of prolactin on fluid and NaCl absorption by the rat proximal and distal colon[1]

J.R. Mainoya[2,3]

Department of Zoology and Cancer Research Laboratory, University of California, Berkeley (California 94720, USA), 14 November 1978

Summary. The rates of fluid and NaCl absorption are greater in the proximal than in the distal colon. Prolactin treatment caused significant increases in fluid and NaCl absorption in the proximal but not in the distal colon. This suggests that only the proximal colon region, where most of the fluid and sodium absorption takes place, is responsive to prolactin.

The mammalian colon fulfills the important function of water and NaCl retention[4-6]. Available information further suggests that the absorption of water and NaCl by the colon is stimulated by raised blood levels of aldosterone and dietary sodium depletion[5,7-11].

Although much of the available evidence indicates that prolactin enhances intestinal absorption of water and ions in the rat intestine[12-16], little is known about the effects of this hormone on the mammalian colon. It is well known that the colon exhibits regional absorptive differences along its length[4,5,7,11,17-19]. However, reports are often at variance with regard to the ability of the proximal and distal colons to transport fluid and NaCl. While some report that most of the NaCl and fluid absorption takes place in the proximal colon[5,11,17,18], others suggest that transport is higher in the distal than in the proximal colon[4,7,19].

In a previous study, we were unable to demonstrate significant increases in mucosal fluid and NaCl transfer in sacs prepared from the entire colon of rats pretreated with prolactin[13]. However, bovine growth hormone and human placental lactogen caused small but significant increases in fluid and NaCl absorption in the rat colon[16].

The present study was undertaken in order to find out what region of the rat colon is responsive to prolactin treatment.

Materials and methods. Male Sprague-Dawley rats weighing 250–300 g were used. The animals were maintained on White diet (Simonsen, Labs, Gilroy, Ca.) and water given ad libitum.

The animals were divided into 2 groups: one group received 1.0 mg prolactin injections and the other group, which acted as control, received the hormone vehicle. Ovine prolactin, (US National Institutes of Health P-S-10: 25.6 IU/mg) was prepared for injection by dissolving 10 mg in 1.0 ml of 0.002 M NaOH and then diluting to 20 ml with

Effect of prolactin (PRL) on fluid and NaCl absorption, and serosal:mucosal ratios of Na^+ and Cl^- concentrations in the rat colon (means ± SEM)

Region of colon	Treatment (number of animals)	Mucosal fluid transfer (ml/g wet wt/h)	Mucosal Na^+ transfer (μEq/g wet wt/h)	Mucosal Cl^- transfer (μEq/g wet wt/h)	Concentration ratio: Na^+ (serosal: mucosal)	Concentration ratio: Cl^- (serosal: mucosal)
Proximal colon	Control (7)	1.27 ± 0.07	267.42 ± 20.57	208.07 ± 17.82	1.23 ± 0.02	1.20 ± 0.02
	1.0 mg PRL (7)	1.83 ± 0.11**	380.57 ± 25.47*	332.48 ± 23.31*	1.22 ± 0.01	1.24 ± 0.01
Distal colon	Control (7)	0.87 ± 0.13	147.81 ± 11.57	182.10 ± 21.87	1.03 ± 0.01	1.20 ± 0.01
	1.0 mg PRL (7)	0.98 ± 0.10	175.62 ± 11.05	201.16 ± 18.47	1.04 ± 0.01	1.20 ± 0.01

Statistical significance of difference from the control group: * $p < 0.01$; ** $p < 0.001$.

0.9% NaCl. Injections were given s.c. in mid-afternoon 48 and 24 h before the animal was used.

From sodium pentobarbitone-anaesthetized rats, the entire colon was removed, rinsed in saline and then everted. 2 5-cm long sacs were then prepared, one from the proximal colon and another from the distal region of the rat colon. Each sac was initially weighed empty using a Mettler H balance, filled with about 1.0 ml of Krebs-Henseleit Ringer solution (pH 7.4) containing 28 mM glucose, and then reweighed.

Each sac was incubated for 1 h in a 125 ml Erlenmeyer flask containing 20 ml of the Ringer solution maintained at 37 °C and gassed continuously with 95% O_2: 5% Co_2 and shaken at 80 oscillations/min.

Following incubation, each sac was reweighed to determine mucosal transfer, and both mucosal and serosal fluids collected for sodium and chloride determinations. Sodium concentrations were determined using a Perkin Elmer atomic absorption spectrophotometer (Model 290B) and chloride concentrations using the Buchler-Cotlove chloridometer.

The transferred amounts were expressed as mucosal fluid or ion transfer/g initial wet weight/h. All values were expressed as means ± SEM and significance was determined by Student's t-test.

Results. From the results, which are summarized in the table, it can be seen that the rates of absorption of fluid and sodium are higher in the proximal than in the distal colon. It is also evident that ovine prolactin administration in rats significantly increased mucosal fluid transfer in the proximal colon ($p < 0.001$) but not in the distal colon. Prolactin also significantly stimulated mucosal sodium transfer in the proximal colon ($p < 0.01$) but again was without significant effect in the distal colon. Similarly mucosal chloride transfer was only significantly increased in the proximal region of the colon ($p < 0.01$). Furthermore it is clear from the table that the sodium concentration ratio of serosal: mucosal (S/M) is > 1 in the proximal colon and ≃ 1 in the distal colon, whereas the (S/M) ratio for chloride is > 1 in both the proximal and distal colons. The concentrations of sodium and chloride in the colon absorbate in the prolactin treated rats remained the same as in the control rats.

Discussion. The results presented in this study are in agreement with the observations of Levitan[4], Dolman and Edmonds[11], but do not support the observations of Hornych et al.[19] who reported that rates of sodium and fluid absorption in the distal colon are higher than in the proximal colon of unoperated control rats.

The most important finding in the present study is the demonstration that prolactin treatment significantly enhanced fluid and NaCl absorption in the proximal colon but had no significant effect in the distal colon.

Although mucosal chloride transfer rates are similar in the proximal and distal colons from control rats, prolactin treatment resulted in greater increases in chloride transfer only in the proximal colon.

It is apparent from the sodium concentration ratios (S/M) that the proximal colon is more actively involved in sodium transport than the distal colon. However, the chloride concentration ratios (S/M) are the same for both colonic regions.

Of the important sodium retaining hormones, aldosterone and angiotensin have been shown to act mainly on the distal colon[7,20]. However, recently evidence has been put forward to the effect that the aldosterone effect is greater in the proximal than in the distal colon[11].

The present observations suggest that prolactin may be one of the important factors regulating fluid and sodium retention by the mammalian colon where it appears to enhance absorption mainly in the proximal region. This action of prolactin may be of greater adaptive significance, especially in lactating mammals which stand to lose substantial amounts of water and salts through suckling their young. Suckling is a very potent stimulus for prolactin secretion[21-23] and the reported increases in the weights of the colon during lactation[24] may be associated with a rise in prolactin secretion[25].

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2 Present address: Department of Zoology, University of Dar es Salaam, P.O. Box 35064, Dar es Salaam (Tanzania).
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