The concentrations of sucrose were the same as used by Cabanac and his group (2.5, 5, 10, 20 and 40% w/v). The solutions were tasted at intervals of 3 min in the same random order before and after the injection. 1 complete series of tasting lasted 12 min. A series of tasting took place immediatly before the injections. The 2nd, post-injection, series of tasting began (in separate experiments) after intervals of either 9, 13, 18 or 36 min. Different subjects were tested for each experiment. Hedonic estimates were made on a 5-point scale from -2 = very unpleasant to $+2$ = very pleasant. Comparisons and statistical calculations were based on differences in scores of whole series of 5 sugar solutions. Scores for each series were obtained by algebraically summing estimates for all 5 solutions in the series. The statistical significance of the differences in hedonic changes (insulin minus saline) was assessed by a 2-tailed matched-pairs signed-ranks Wilcoxon test. Only 1 condition yielded statistically significant results: the taste of sugar solutions was rated as more pleasant 36–48 min after the insulin injection than after the control injection ($N=32$; $T=137$, $z=2.38$, $p<0.02$). In this experiment 38 subjects were tested; in 6 of them no differences were found between the changes in the hedonic estimates produced by insulin and those produced by saline. When post-injection series of tasting started at 9, 13 or 18 min after insulin, no significant differences were found with control experiments. In some subjects, blood sugar level was determined before and after the last series of tasting both after insulin and after saline injections.

Possible correlations of blood sugar levels with changes in hedonic estimates were assessed. In only 2 cases were correlations at the limit of statistical significance found, as shown in the figure.

In those 2 distinct experiments, the hedonic changes induced by insulin correlated negatively ($r=-0.595$) with blood sugar at 30 min and positively ($r=0.416$) with blood sugar at 50 min. In other words, in 1 experiment the lower the blood sugar at 30 min after the insulin, the better did the 5 solutions taste between 36 and 48 min; while, on the contrary, in the other experiment, the higher the blood sugar at 50 min, the better the sugar solutions tasted. These opposite correlations of alliesthetic changes with blood sugar at 30 and 50 min can be associated with the findings of Grossman and Stein[3], Janowitz and Ivy[4] and Silverstone and Besser[5], that hunger after insulin begins after the depth of hypoglycemia has passed.

Since no negative alliesthesia was found, exogenous insulin is unlikely to be a satiety signal, at least with the dose used and at the times explored. On the other hand, the positive alliesthesia found does not imply that insulin is a physiological hunger signal, as there are many arguments against this view. Rather, we may be dealing with a not very reliable emergency response.

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Liver vitamin A stores in chronic alcoholism in rats: Effect of propylthiouracil treatment

G.D. Nadkarni, U.R. Deshpande and D.N. Pahuja

Radiation Medicine Centre, Medical Group, B.A.R.C., Tata Memorial Centre Annexe, Jerbai Wadia Road, Parel, Bombay 400 012 (India), 1 November 1978

Summary. Administration of alcohol to rats through drinking water for 8 weeks produced a significant decrease in the liver vitamin A stores without causing any change in the plasma vitamin A levels. Treatment of the alcoholic rats with propylthiouracil for 2 weeks restored the liver vitamin A reserves to control levels.

The liver vitamin A stores are comparatively stable in animals which are not on vitamin A-deficient diet[1]. There are very few reports[2–4] on the effect of short-term and chronic alcohol administration on liver vitamin A. We report here our studies on this parameter in rats on normal diet and those receiving alcohol in drinking water for 8 weeks. We further report the effect of propylthiouracil (PTU) treatment of chronically alcoholic rats on liver vitamin A, since PTU is known to protect the liver from alcohol injury[5]. Our findings support the protective role of PTU in alcohol-induced depletion of vitamin A stores of the liver.

Material and methods. 3 groups of male Wistar rats, weighing 100–120 g, were put on our standard laboratory diet of 20% protein, consisting of cracked wheat, powdered Bengal gram, dry fish meal, yeast powder, shark liver oil and sesame oil. 2 groups of rats were made chronically alcoholic as described by Pritchard and Schneck[6], except that the final concentration of alcohol was 10% in 10% sucrose from 5th week onwards. 1 group acted as control and received only 10% sucrose in tap water for drinking. Based on the daily consumption of drinking water, it was estimated that the alcohol intake was approximately 5g/kg/day when on 10% alcohol regimen.

After 6 weeks on alcohol, 1 of the groups started receiving 5mg/kg/day of PTU in drinking water along with 10% alcohol for 2 weeks, while the 2nd group received only alcohol. 8 weeks after the commencement of the experiment, each rat in all the groups received approximately 20 IU of 2.5 μCi retinyl-11-12-^{3}H-acetate (by courtesy of Hoffman-La Roche Inc.) and the liver vitamin A stores were estimated by the method proposed by Bausch and Rietz[7]. Vitamin A in the plasma was estimated by a fluorimetric method using a modification of the methods of Selvaraj and Susheela[8] and Thompson et al.[9].

Results and discussion. A recently proposed indirect method for the assessment of vitamin A liver stores[7] has helped us to study the chronic effect of alcohol on this parameter.

Plasma and liver vitamin A concentrations

Group	Plasma vitamin A (IU/100 ml)	Liver vitamin A (IU/g)
Control (8)	176 ± 32	119 ± 13
Alcohol (8)	168 ± 26	86 ± 12*
Alcohol + PTU (7)	192 ± 68	135 ± 24

Values are mean ± SD for number of rats shown in parenthesis.
* Significantly different from controls, $p<0.001$.

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