Hyperactivities of the thyroid and adrenal glands has been shown to occur in ascorbic acid deficiency[16]. Recently Clenano et al.[17] demonstrated that cortisone or tri-iodothyronine injections to female rats can elicit the precocious appearance of the disaccharidases in intestine. Therefore the changes observed in the digestive functions of the small intestine could possibly be related to the hormonal imbalances associated with vitamin C deficiency. There is ample kinetic evidence which emphasizes a close functional relationship between the brush border disaccharidases and the sugar transport system at the mucosal surface of the enterocytes[18,19]. The similarities observed in the alterations of brush border sucrase and glucose absorption in intestine in vitamin C deficiency and after feeding excessive doses of the vitamin, suggests a common control mechanism for these systems under the 2 nutritional statuses. The results of the present study provide indirect evidence for such a parallelism between the 2 systems.

Whether ascorbic acid is directly involved in the intestinal digestive and absorptive mechanisms, or is linked to these processes indirectly by inducing metabolic alterations, is not clear at present; nevertheless the hyperactivities of the brush border enzymes and the enhanced permeability of sugars and amino acids in intestine in scurvy seems to be a characteristic of this derangement. Excessive intake of vitamin C as an antidote for common cold and other ailments has often been advocated[20]. In view of the observed changes in the intestinal functions after feeding large doses of vitamin C, such a vitamin therapy needs a further careful exploration.

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Positive alliesthesia after insulin

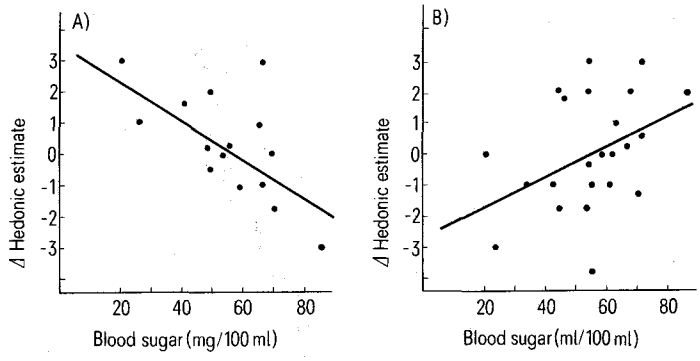
E. Briese and M. Quijada

Behavior Physiology Laboratory, Universidad de Los Andes, Apartado 109, Mérida (Venezuela), 23 October 1978

Summary. Volunteers experienced sucrose solution as more pleasant 36–48 min after insulin, than after saline control. These changes in affective estimates correlate negatively with blood sugar at 30 min and positively at 50 min after the insulin injection.

The same stimuli can be perceived as pleasant or unpleasant according to the internal state, that is, the homeostatic situation. This phenomenon has been called by Cabanac[1] alliesthesia and plays an important role in the reinforcement of the behavior and behavioral regulation of some homeostatic functions. For instance, in the case of olfactogustative stimuli, the alliesthesia parallels hunger and satiety. Since, inspite of being often investigated the role of insulin in hunger and satiety is not yet clear[2], and because insulin is a major metabolic hormone supposed to influence homeostatic energetic balance, we wanted to see if insulin produces some changes in the evaluation of the taste of sucrose.

In double-blind experiments, 85 healthy young volunteers were asked, in the morning before breakfest, to rate the pleasantness or unpleasantness which they felt about the taste of 5 sucrose solutions before and after an i.m. injection of 0.15 units/kg wt of normal insulin or isotonic saline.



Correlations between the blood sugar 30 min (A) and 50 min (B) after i.m. injection of 0.15 units/kg b.wt and changes in hedonic estimates of sucrose solutions tasted 36–48 min after the injection. Each point represents the differences between the affective estimates before and after the injection. *A* $y = -0.059\ x + 3.610$, $r = -0.595$, $p < 0.02$; *B* $y = 0.048\ x - 2.67$, $r = 0.416$, $p < 0.05$.

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, $p<0.001$.