slightly opalescent. Nevertheless, complete inhibition was achieved only with $10^{-3}M$ suggesting that the concentration of the active form, which probably is the free cation, kept increasing beyond this point. The concentrations for 50% inhibition in these 2 experiments were $0.93\times10^{-4}M$ for Ho and $1.45\times10^{-4}M$ for Pr. Figure 2 demonstrates that failure to extrude Ca under the influence of the lanthanides reflects an interference with the transport mechanism and is not due to leakage of Ca from the cells. In this experiment an unusually large gradient of Ca from inside to outside was set up. It can be seen that this gradient remains nearly constant during 30 min in the cells poisoned with Ho, whereas in the control sample Ca leaves the cells in a downhill direction.

Figure 3A shows concentration-effect curves for the inhibition by Ho of the part of the Mg + Ca requiring ATPase not dependent on Na or K. The curves differ in position relative to the Ho concentration axis by less than a factor of two when the Ca concentration is varied by a factor of 10. At Ca 0.06 mM the apparent K_m for Ho was $0.67\times10^{-4}M$ and at Ca 0.6 mM it was $0.87\times10^{-4}M$ in this experiment. These values indicate that the affinity for Ho of the particular Ca + Mg activated ATPase system in the membrane and of the Ca transport system are quite similar. Since full inhibition is obtained with 1 mM Ho the possibility can be ruled out that complex formation with ATP, present in 2 mM concentration, is the cause. The parallel displacement to the right of the curves by Ca is compatible with, but does not conclusively prove competition between Ca and Ho for some site which, when occupied by Ca, is activated. The range of Ca concentrations accessible for such studies is narrow because the activation curve for Ca does not reach a plateau but declines at concentrations above 0.3–0.5 mM. Figure 3B shows that increasing the Mg-concentration, rather surprisingly, shifts the curve to the left.

In summary the 2 three-valent lanthanides tested inhibit both the active uphill Ca transport from resealed red cells and a membrane ATPase activated by Ca which, at least in part, might be involved in this transport. However, as can be learned from the experiment shown in the Table, other ATPases of the membrane, including the classical Na + K stimulated fraction, are inhibited as well. The enzymic activity requiring only Mg seems not to be affected even by high concentrations of Ho^{3+}. The insensitivity of the Mg-ATPase and the fact that Mg does not counteract but enhances the action of Ho^{3+} on the Ca + Mg activated ATPase (Figure 3B) suggest that the Mg-ATP accepting site in the membrane is not a likely point of attack of Ho^{3+}. It might be possible that the lanthanides interfere with the site specific for Ca. This assumption, however, leaves the question open as to why the Mg + Na + K activated ATPase is also affected.

The present finding affords, if nothing else, a convenient experimental tool to block the Ca transport mechanism without interferring with the passive permeability of the membrane for Ca.

Zusammenfassung. Die Lanthanide Holmium^{III} und Praseodymium^{III} hemmen den aktiven Ca-Transport der Erythrozytenmembran. Eine Konzentration von etwa $10^{-4}M$ ergibt 50% Hemmung. Eine Ca + Mg aktivierte Membran-ATPase des Erythrozyten zeigt gegenüber diesen beiden Kationen ungefähr die gleiche Empfindlichkeit wie der Transport.

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Changes in Intestinal Absorption of Glucose in Rats Treated with Ethanol

The majority of the clinical and experimental investigations which have been carried out so far on alcoholism are concerned with structural, functional and metabolic alterations elicited by ethanol in the liver[1-5], cardiocirculatory system[6,7] and central nervous system[8,9]. Conversely, little is known about possible variations in the intestinal absorption process which are brought about by ethanol. The purpose of the research project described in this paper was to ascertain whether subacute oral ethanol poisoning gives rise to alterations of glucose intestinal absorption in rats.

Materials and methods. The investigation was carried out on male albino rats of the Sprague-Dawley strain, weighing on average 180 g. The animals were divided into 4 groups. The rats in the first group were not treated with ethanol and were used as controls. The rats in the 2nd, 3rd and 4th groups were pre-treated with 40% ethanol for periods of 2, 7 and 20 days respectively. The alcohol was administered by gastric tube at a dose of 4 g/kg/day. All the rats were fed ad libitum. The experiment was carried out after 5 h fasting in the rats of the first group, and 5 h after the last ethanol administration in those of the 2nd, 3rd and 4th groups which had also fasted for 5 h.

The rats were anaesthetized with ethylurethane (1 g/kg i.p.) and then laparatomized. The small intestine was closed at the level of the duodenum and the ileum-cecal valve; 5 ml of a 20% solution of pure glucose were injected into the intestine. Each animal was re-opened 1 h after glucose administration. The small intestine, approximately 2 ml of aortic blood, and about 500 mg of liver tissue were taken from every animal.

Glycemia was determined in the blood by the Hugget and Nixon[10] enzymatic method.

Residual glucose in small intestine. The small intestine was washed and the washing water added to the contents

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Glucose absorbed by small intestine, glycemia and liver glycogen in control rats, and in rats treated with ethanol (4 g/kg/die per os, 40% solution) for 2, 7 and 20 days

	No. of rats	Glucose absorbed by small intestine (mg/100 cm/h)	Glycemia (g of glucose/ 1000 ml of plasma)	Liver glycogen (g of glucose/ 100 g of wet liver)
Control rats	14	357 ± 17	6.70 ± 0.32	0.41 ± 0.08
Rats treated with ethanol for 2 days	14	294 ± 10^{b}	8.14 ± 0.20^{b}	0.67 ± 0.23
Rats treated with ethanol for 7 days	14	294 ± 60^{a}	8.87 ± 0.49^{b}	0.66 ± 0.26
Rats treated with ethanol for 20 days	14	349 ± 29	7.84 ± 0.39^{a}	0.59 ± 0.21

1 g of glucose was administered in the small intestine of all the rats. Mean values $\pm$ S.E. ^{a} $0.05 \geqslant P \geqslant 0.02$. ^{b} $0.01 \geqslant P \geqslant 0.001$.

of the small intestine. The whole mass was brought to 100 ml volume. The solution was then diluted 50 times and centrifuged. Glucose was determined in the supernatant by the HUGGET and NIXON[10] enzymatic method.

Liver glycogen expressed as glucose. The 500 mg of liver tissue taken from the live animals were added to 3 ml of 30% KOH and kept in a water-bath at 100 °C for 30 min. Glycogen was precipitated by adding 95% ethanol. It was washed with 95% ethanol and then hydrolyzed to glucose with 10 ml of $1N$ H_2SO_4 in a water-bath at 100 °C for 4 h. The mixture was neutralized with $1N$ NaOH and brought to 25 ml volume. Glucose was determined in this solution by the HUGGET and NIXON[10] enzymatic method.

Results. As shown in the Table, intestinal absorption of glucose decreases by 18% in rats treated with ethanol for 2 and 7 days, whereas it reaches almost similar values to those of the control rats in animals treated with ethanol for 20 days. As compared with the controls, glycemia increases by 21%, 32% and 17% in rats treated with ethanol for 2, 7 and 20 days respectively. Liver glycogen does not undergo statistically significant variations. Its values are very low and irregular, probably as a result of the 5 h fasting undergone by the rats before the experiment.

Discussion. The glycemia increase in rats poisoned with ethanol, in comparison with the control animals, could be caused by competitive inhibition of glucose utilization by ethanol and FFA[11]. In effect, it is known that there is an increase in plasmatic FFA caused by alcohol intoxication[2-4,12]. Intestinal absorption of glucose occurs in 2 stages: the first by means of active transport by an enzymatic mechanism, and the second by simple diffusion[13]. The enzymes which regulate the first stage of glucose absorption could either be directly or indirectly inhibited by ethanol. On the other hand, the highest glycemia levels observed in the animals treated with ethanol can slow down the diffusing process of glucose. both assumptions can explain the decrease in intestinal absorption of glucose which was observed in the rats poisoned with ethanol. Changes in glycemia and glucose intestinal absorption reached a maximum after 2 and 7 days of ethanol treatment and were less marked or even absent after 20 days of alcoholic intoxication.

The results show that the rat possesses a mechanism of ethanol inurement which is induced with time. This assumption of ours is supported by the findings of ALBERTINI et al.[14]. These authors observed that the liver alcoholdehydrogenase activity of the rat increases considerably due to both acute and chronic ethanol poisoning. Our data allow us to draw the conclusion that the changes observed both in glycemia and in intestinal absorption of glucose can be ascribed to an acute or subacute rather than to a chronic effect of alcoholic intoxication.

Riassunto. Gli Autori hanno indagato se l'intossicazione subacuta da etanolo provoca nel ratto alterazioni dell'assorbimento intestinale del glucosio. L'assorbimento intestinale del glucosio è diminuito in maniera statisticamente significativa nei ratti trattati per 2 e 7 giorni ed è risultato quasi normale in quelli trattati per 20 giorni con etanolo.

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Native Fluorescence and Hallucinogenic Potency of Some Amphetamines

The native fluorescence of a molecule is a complex function of its quantum chemical properties that may include the energy of its π electrons. SNYDER and MERRILL[1], and KANG and GREEN[2] have reported that the HMO energy of methoxylated amphetamines correlates positively with their hallucinogenic potency. We have, therefore, measured the native fluorescence, at maximum activation and emission frequencies, of a number of amphetamines to see if there was any correlation with their hallucinogenic potency (as measured by SHULGIN et al.[3]). This work was carried out independently in Iowa and Edinburgh.

Measurements were taken with spectrophotofluorometer using amounts of the compound (as hydrochloride) isomolar to 10 µg of amphetamine HCl in 2 ml water volume. The degree of fluorescence is expressed in the direct readings from the Farrand (Edinburgh) and Aminco – Bowman (Iowa) and the biological activity is expressed in mescaline units, i.e. how many times the compound is as potent as mescaline as measured by SHULGIN et al.[3].

The relative intensity of emission was calibrated in the results from the 2 centres by multiplying each of the

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