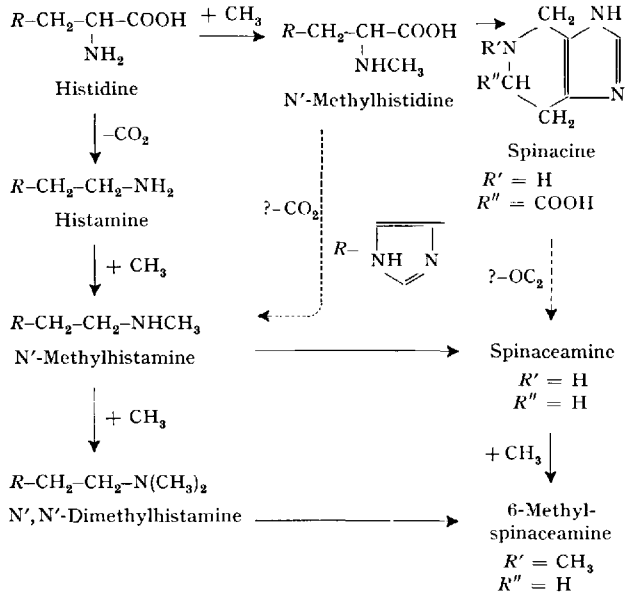
The approximate content in imidazoalkylamines of the dry skin obtained from 5 specimens of *Leptodactylus pentadactylus labyrinthicus* captured in Misiones in September 1961 was as follows (in μg of free bases per g dry tissue): histamine 360–400, N'-methylhistamine 250–300, N',N'-dimethylhistamine 100–120, spinaceamine 60–70, 6-methylspinaceamine 200–220.



The possible biochemical correlations among the different imidazolealkylamines of the *Leptodactylus* skin are illustrated below.

Details on methods and data obtained in other *Leptodactylus pentadactylus* species as well as in other amphibians will be presented in the paper *in extenso*, together with a full discussion of results.

Riassunto. Estratti di pelle di *Leptodactylus pentadactylus labyrinthicus* contengono elevati quantitativi di imidazolalchilamine, fra cui due derivati imidazo-*c*-piridinici finora ignoti in natura: la spinaceamina e la 6-metilspinaceamina.

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The Sodium and Glucose Transports by *in vitro* Intestinal Preparations[1]

The experimental data reported here have been obtained on isolated everted sacs of small intestine of albino rat prepared according to Wilson and Wiseman[2]. As incubating mucosal fluid, the Krebs–Henseleit[3] solution or a modified one (see Tables) was used. The intestinal sacs were incubated for 1 h and 30 min at 38°C or at 28°C; a low temperature seems to improve the efficiency of the preparation. The volume of the serosal fluid at the end of the incubation period was determined by weighing. The fresh weight of the emptied intestinal sac was also determined at the end of the experiment.

The glucose concentrations in the mucosal and serosal fluids were determined at the end of the experiment according to the method of King[4]; from these concentrations, the glucose which disappears in the mucosal fluid, and the glucose which appears in the serosal fluid, were calculated. The sodium concentration in the serosal fluid was determined at the end of the experiment by means of an 'Optica CF 4' flame spectrophotometer (λ = 589 mμ); from this concentration the sodium gain of the serosal medium (net sodium transfer) was calculated. The lactic acid concentrations in the mucosal and serosal fluids were determined at the end of the experiment according to the method of Barker and Summerson[5]; from these concentrations the whole production of lactic acid was calculated. The glucose and lactic acid determinations allow us to define the following glucose quantities: glucose absorption = glucose disappearing in the mucosal fluid; glucose transfer = glucose appearing in the serosal fluid; glucose utilization = glucose absorption minus glucose transfer; unrecovered glucose = absorbed glucose minus transferred glucose minus glucose appearing as lactic acid.

In a previous report Rossi et al.[6] emphasize the fact that the utilization of glucose by the isolated perfused intestine, according to the technique of Smyth and Taylor[7], seems not to be dependent on the sodium concentration in the mucosal fluid, when NaCl is replaced by isosmolar quantities of urea or mannitol; on the contrary, the glucose transfer seems to be correlated with the net sodium transfer and with the sodium concentration in the mucosal fluid.

Present observations substantially confirm the previous ones. If the values of net sodium transfer obtained in control experiments are plotted against the values of glucose absorption (Figure, A) or of glucose transfer (Figure, B), a correlation appears in the second case. A correlation between net sodium transfer and unrecovered glucose cannot even be detected. In the experiments in which 2,4-dinitrophenol (DNP) was used (Table I), in spite of the marked inhibition of the sodium pump, there is little change in glucose absorption, in accordance with similar observations of Matthews and Smyth[8] and Lippe et al.[9], and the glucose transfer is completely abolished. Significant differences in the production of lactic acid do not appear under these experimental conditions, so that the change in glucose absorption may be simply

[1] This work has been supported by a research grant of the Consiglio Nazionale delle Ricerche, Roma.
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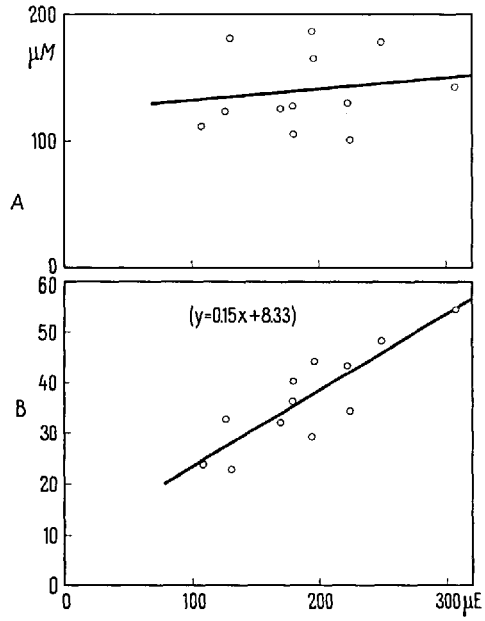
Table I. Everted small intestine of albino male rat (Wistar strain) weighing about 250 g. Jejunum portions starting 20 cm from pylorus and weighing about 0.5/1 g were chosen. In each everted sac was initially introduced 0.3 ml of the Krebs solution without glucose and the sac was then incubated throughout 1 h and 30 min in the same Krebs solution or a modified one (see Table) containing 13.9 mM/l of glucose. Initial quantity of incubating solution: 50 ml. Oxygenation with a mixture of O_2 95% and CO_2 5%. The number of experiments (n) for each group and the mean values ± S.E., referred to 1 g fresh weight and 1 h, are reported

Incubating mucosal fluid	Glucose absorbed $\mu M\ g^{-1}\ h^{-1}$	Na net transfer $\mu E\ g^{-1}\ h^{-1}$	Glucose transfer $\mu M\ g^{-1}\ h^{-1}$	Lactic acid produced $\mu M\ g^{-1}\ h^{-1}$
	Temperature of incubation 38°C			
Na conc. 143.5 mE/l n = 8	192.5 ± 12.3	138.0 ± 11.9	21.2 ± 2.9	
Na conc. 54 mE/l NaCl partially replaced by isosmolar urea n = 7	184.0 ± 9.9	60.0 ± 9.0	10.6 ± 3.3	
Na conc. 143.5 mE/l DNP 0.1 mM/l n = 8	136.9 ± 19.6	50.5 ± 4.8	0.9 ± 0.5	
	Temperature of incubation 28°C			
Na conc. 143,5 mE/l n = 12	140.5 ± 8.8	190.5 ± 16.2	36.5 ± 2.3	145.0 ± 10.3
Na conc. 54 mE/l NaCl partially replaced by isosmolar urea n = 8	82.1 ± 9.6	50.1 ± 9.1 [Na active influx 110 μE]	11.8 ± 3.7	117.8 ± 5.5
Na conc. 143.5 mE/l DNP 0.1 mM/l n = 7	110.5 ± 22.0	58.4 ± 7.3	0.8 ± 0.5	138.0 ± 16.2

due to the absence of glucose transfer and the unrecovered glucose seems not to differ from that in control experiments. Furthermore, when the net sodium transfer is reduced by lowering the sodium concentration in the mucosal fluid (urea experiments, Table I), the glucose transfer is affected much more strongly than the glucose absorption. The decrease of glucose absorption observed in urea experiments at 28°C, may be related to the concomitance of low temperature and low sodium concentration *per se*, as admitted by BIHLER, HAWKINS and CRANE[10], without depending on the sodium pump.

In conclusion, the existence of a correlation between glucose and sodium transfers is well established, whereas the absorption of glucose by the intestinal epithelium seems not to be affected by the net sodium transfer, under our conditions. The glucose transfer is higher in urea experiments than in DNP experiments, in spite of a similar net sodium transfer (Table I). An interference of DNP with the glucose transfer, more pronounced than that with the sodium net transfer, may be an obvious explanation of the above dissociation between glucose and sodium transfers (LIPPE et al.[9]). However, the following possibility must also be taken into account. The net sodium gain of the serosal medium may represent quantitatively the activity of the sodium pump only if the intestine is incubated with fluids containing at both sides an equal sodium concentration. On the contrary, the net sodium gain may be noticeably inferior to the value of the sodium pump, if the intestine is incubated in a mucosal fluid with a sodium concentration lower than the one of the serosal fluid, because of the passive back diffusion of sodium from the serosal to the mucosal side.

In order to verify such a possibility, the total sodium flux from the serosal to the mucosal medium (sodium outflux) has been determined in four intestinal sacs incubated in Krebs fluids, in four intestinal sacs poisoned with DNP, and in four intestinal sacs incubated in a mucosal fluid in which NaCl of the Krebs solution was partially replaced by urea. The experimental data so far collected are reported in Table II. From these data the conclusion may be drawn that the sodium outflux is a diffusional flux practically independent of the sodium pump. Similar results are reported also by CURRAN[11].



Abscissa: Na net transport $\mu E\ g^{-1}h^{-1}$. Ordinata: (A) glucose absorption $\mu M\ g^{-1}h^{-1}$, (B) glucose transfer $\mu M\ g^{-1}h^{-1}$.

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Table II. Everted small intestine of albino male rat (Wistar strain) weighing about 250 g. Jejunum portions starting 20 cm from pylorus and weighing about 0.5/1 g were chosen. In each everted sac 1 ml of the Krebs solution without glucose and containing $^{24}NaCl$ [a] (total c.p.m. 10^4–$2 \cdot 10^4$), was initially introduced and the sac was then incubated throughout 1 h 30 min in 50 ml of the Krebs solution or of a modified one (see Table) containing 13.9 mM/l of glucose. The radioactivity appearing in this fluid was determined at the end of the experiment. Temperature of incubation 28°C. Oxygenation with a mixture of O_2 95% and CO_2 5%. The single values per g fresh weight and per h are reported

Incubating mucosal fluid	Mean serosal Sodium conc. mE/l	Sodium total outflow $\mu E\ g^{-1}\ h^{-1}$
Krebs + glucose	134.5	141.3
13.9 mM/l	135.5	158.5
Na conc. 143.5 mE/l	135.5	152.0
	135.0	157.2
	m = 135.1	m = 152.2
Krebs + glucose	123.0	116.5
13.9 mM/l	118.0	126.4
Na conc. 54 mE/l	117.0	107.5
NaCl partially replaced by isosmotic urea	114.5	148.5
	m = 118.1	m = 124.7
Krebs + glucose	137.0	159.4
13.9 mM/l	139.0	138.5
Na conc. 143.5 mE/l	138.5	156.4
DNP 0.1 mM/l	134.5	137.0
	m = 137.2	m = 147.8

[a] Kindly supplied by Centro Studi Nucleari E. Fermi, Milano.

Presumably there exist diffusional fluxes (outflux and influx) which cross the intestinal wall in sites different from those utilized by the sodium pump. If such an assumption is correct, one may attempt to calculate the active sodium influx (ϕ_i^a) by the following formula:

$$\phi_i^a = \Delta\mathrm{Na} + \phi_o^d - \phi_i^d$$

where (ϕ_o^d) and (ϕ_i^d) are the diffusional out- and influxes, which are tentatively assumed to be only proportional to the serosal, respectively mucosal, sodium concentration, and (ΔNa) is the net sodium gain of the serosal medium. By introducing in the above equation the mean value of (ΔNa) determined in the urea experiments (Table I) and the corresponding values of (ϕ_o^d) and (ϕ_i^d) calculated on the basis of the experimental mean value of (ϕ_o^d) (reported in Table II: 124.7 $\mu E\ g^{-1}\ h^{-1}$), an active sodium influx of 110 $\mu E\ g^{-1}\ h^{-1}$ has been obtained:

$$\phi_i^a = 50.1 + \frac{110.5 \cdot 124.7}{118.1} - \frac{54 \cdot 124.7}{118.1} \cong 110\ \mu E\ g^{-1}\ h^{-1}$$

This flux (Table I, in brackets) is clearly higher than the net sodium transfer; with the lowering of the sodium pump the glucose transfer decreases more strikingly and the behaviour of the intestine poisoned with DNP seems no longer to disagree with that observed in urea experiments. In our opinion, this correlation between sodium and glucose transfers, besides signifying that the presence of a definite sodium concentration into the epithelial cells is necessary for the functioning of the sugar transfer, as supposed by CSÁKY[12], means that the sodium pump itself must function so that the glucose transfer may take place.

Riassunto. L'assorbimento di glucosio da parte del digiuno di ratto, rovesciato ed incubato *in vitro*, appare relativamente poco sensibile al trasporto attivo del sodio, mentre il trasporto di glucosio dalla mucosa alla sierosa è fortemente correlato e nello stesso senso, con il trasporto attivo di sodio.

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Galactose Intoxication Pathologic Study in the Chick[1]

LECOQ et al.[2] in 1943 reported that pigeons on a diet containing 66% galactose developed symptoms similar to vitamin B_1 deficiency. DAM[3] observed that young chicks fed a 55% galactose diet developed convulsions and died after several days. In 1953 RUTTER et al.[4] found that chicks tolerated galactose up to 10% but greater amounts resulted in ataxia, tremors and eliptiform convulsions. A purified diet containing 10% galactose was fed to chicks by FOX and BRIGGS[5]. The authors reported no observable change from chicks fed the basal diet, and no curled toes or convulsions were found in the galactose-fed chicks. No pathologic lesions have been reported in

[1] Supported by grant B 2951 (C1) from the National Institute of Neurological Diseases and Blindness, National Institutes of Health, United States Public Health Service.

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