treated preparations. However, because some of the supporting structure had been removed by enzyme treatment, these bundles were easily damaged.

Results of the microelectrode studies are presented in the table and the figure. Although a slight reduction was noted in the resting potential with the enzymetreated muscles, the action potential overshoot of the isoelectric line and the action potential amplitude were not significantly ($p < 0.05$) different from the untreated preparations.

Discussion. Proteolytic enzymes such as trypsin have been shown to have deleterious effects on skeletal muscle transmembrane potentials[5]. Collagenases are more specific in cleaving primarily intercellular proteins and have been used to recover viable cells from a variety of tissues[1,2,6]. In the experiments reported here, we applied collagenase to rat skeletal muscle using a hind-limp perfusion technique to facilitate enzyme distribution and subsequent fibre bundle dispersion. The results indicate that collagenase pretreatment has only a slight, or negligible, effect on the cellular resting potential and action potential magnitudes. Action potential durations were observed in some preparations to become prolonged. However, this occurred only when the action potential and resting potential amplitudes began to decay. We feel this technique offers a simple adjunct to mammalian skeletal muscle dissection, and thus may be particularly useful in studies of electrophysical and thermomechanical phenomena.

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The effect of dehydration on the neurohypophyseal blood flow in rats with hereditary diabetes insipidus[1]

J. Kapitola[2], H. Dlouhá, J. Křeček[3] and J. Zicha

Institute of Physiology, Czechoslovak Academy of Sciences, Krč Budějovická 1083, CS-142 20 Praha 4 (Czechoslovakia), 25 May 1977

Summary. Neurohypophyseal blood flow increases in water-deprived rats. This increase is independent of vasopressin release, since it occurs even in rats with hereditary defect of hypothalamic vasopressin synthesis.

Using ^{125}I-antipyrin, Lichardus et al.[4] found an increase of neurohypophyseal blood flow in rats after water deprivation, and they concluded that it accompanied the release of vasopressin (VP). Earlier it was reported that any stimulation of neurohypophyseal hormones release causes a vasodilatation in posterior pituitary[5]. Furthermore, water deprivation causes the release, from the neurohypophysis, of both oxytocin and VP[6]. An attempt is presented to ascertain whether neurohypophyseal blood flow increases after water deprivation even in rats which are unable to synthetize VP, i.e. in homozygous Brattleboro rats with hereditary diabetes insipidus[7].

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2 Laboratory for Endocrinology and Metabolism, Faculty of General Medicine, Charles University, Prague.
3 Reprint requests: Dr J. Křeček, Institute of Physiology, Czechoslovak Academy of Sciences, 14220, Prague 4.
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Body weight, adenohypophyseal and neurohypophyseal weight, flow fraction of cardiac output and ^{86}Rb/g-uptake in control (C) and water deprived (WD) Wistar, heterozygous (non-DI) and homozygous (DI) Brattleboro male rats

	Wistar		Non-DI		DI	
	C	WD	C	WD	C	WD
Body weight (g)	204.4 ± 5.56 (9)	187.5[a] ± 2.50 (8)	247.1 ± 7.47 (7)	216.3[a] ± 6.25 (8)	190.7 ± 7.35 (7)	160.6[a] ± 5.21 (8)
Adenohypophysis						
Weight (mg/100 g b.wt)	–	–	1.98 ± 0.061	1.93 ± 0.071	2.05 ± 0.061	2.29[c] ± 0.108
Flow fraction of cardiac output ($\% \times 10^{-3}$)	–	–	2.49 ± 0.257	2.31 ± 0.206	2.74 ± 0.229	3.04[c] ± 0.175
^{86}Rb-uptake (%/g)	–	–	0.62 ± 0.056	0.60 ± 0.046	0.67 ± 0.048	0.67 ± 0.046
Neurohypophysis						
Weight (mg/100 g b.wt)	0.52 ± 0.026	0.55 ± 0.027	0.43[b] ± 0.027	0.48 ± 0.022	0.68[bc] ± 0.034	0.81[abc] ± 0.016
Flow fraction of cardiac output ($\% \times 10^{-3}$)	4.09 ± 0.268	5.04[a] ± 0.352	3.90 ± 0.350	4.71 ± 0.212	4.92[bc] ± 0.256	7.53[abc] ± 0.657
^{86}Rb-uptake (%/g)	3.94 ± 0.243	4.58[a] ± 0.167	4.55 ± 0.354	4.90 ± 0.210	3.67 ± 0.229	4.68[a] ± 0.390

Data are given as mean ± SEM ( ) indicates number of animals.
[a] significantly different from control animals ($p<0.05$); [b] significantly different from Wistar rats ($p<0.05$); [c] significantly different from non-DI rats ($p<0.05$).

Materials and methods. 17 males of Wistar strain, 15 homozygous (DI) and 15 heterozygous (non-DI) male rats of Brattleboro strain aged 90 days and reared from birth under standard laboratory conditions were used. 8 animals in each group were deprived of water 24 h before the onset of the experiment. Tissue ^{86}Rb-uptake was used as an indicator of local tissue blood flow[8] as modified for unanaesthetized animals[9]. The ^{86}Rb-distribution in the course of the first min after i.v. injection corresponds to cardiac output distribution. 20 μCi ^{86}Rb in the form of RbCl (Isocommertz, GDR) were injected into the tail vein and 40 sec later the rat was decapitated. The activity of samples and of ^{86}Rb standard was measured by Autowell II (Picker, USA). According to Sapirstein[8], tissue ^{86}Rb-content was expressed in percent of total administered dose (organ flow fraction in percent of cardiac output) and in percent of total ^{86}Rb-dose per g tissue (corrected for a 'standard rat' weighing 200 g). ^{86}Rb-uptake was measured in anterior and posterior pituitary separately and for comparison also in heart, kidneys and in samples of liver, skin, intestine and muscle. Data were statistically processed by means of Student's t-test.

Results and discussion. In accordance with earlier observations[5], no changes were observed after water deprivation either in DI or in non-DI rats in anterior pituitary weight, flow fraction and ^{86}Rb/g uptake. On the other hand, there was a significant increase in flow fraction of cardiac output and in ^{86}Rb/g uptake in neurohypophysis of DI and Wistar rats. The increase of corresponding values was not significant in non-DI rats. In agreement with data reported by Sokol and Valtin[10], heavier neurohypophysis were found in DI than in non-DI and Wistar rats, and this weight further increased after water deprivation only in DI rats. Dehydration did not influence ^{86}Rb/g-uptake in other organs studied in non-DI rats, while in DI rats ^{86}Rb/g-uptake was decreased in intestine (0.95 ± 0.109 vs. 0.41 ± 0.049) and in skin (0.17 ± 0.016 vs. 0.12 ± 0.010). After water deprivation, the weight of myocardium increased in DI rats (316.3 ± 5.7 vs. 333.0 ± 4.6 mg/100 g b.wt).

An increase of the neurohypophyseal flow fraction of cardiac output and of ^{86}Rb/g-uptake was observed after osmotic load produced by 24 h of water deprivation of Wistar males. But the increase of both parameters was much more expressed in homozygous Brattleboro rats although they do not synthetize VP. This increase need not be in relation to the synthesis or release of VP, and it might be related to the oxytocin release[6], the turnover of which seems to be enhanced in DI rats[11].

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Vitamin C and gallstone formation: A preliminary report

S. A. Jenkins[1]

Pye Research Centre, Walnut Tree Manor, Haughley Green (Suffolk IP14 3RS, England), 9 June 1977

Summary. Gallstone formation in hypovitaminotic C guinea-pigs fed a high cholesterol diet was associated with qualitative changes in the gallbladder bile, namely, a high cholesterol concentration, a lowered bile acid content and diminished phospholipid-to-cholesterol and bile acid-to-cholesterol ratio.

The formation of cholesterol gallstones has been induced experimentally in several species of animal by modifications to their diet[2–6]. Although experimentally induced vitamin A- and to a lesser extent vitamin D-deficiency have been shown to promote gallstone formation[7], no evidence for a relationship between vitamin C status and gallstone formation has appeared. This is perhaps surprizing in view of the considerable amount of experimental evidence linking vitamin C with cholesterol metabolism[8–12]. During the course of a wide-ranging study of the effects of atherogenic diets in guinea-pigs, a high incidence of gallstone formation was observed in animals with latent vitamin C-deficiency. We report here on an initial experiment designed to examine the relationships between vitamin C and gallstone formation.

Methods. 36 male guinea-pigs (Dunkin Hartley) weighing approximately 200 g were given access to water and a standard pelleted laboratory diet ad libitum for 2 weeks, and then transferred to a pelleted high cholesterol (0.5%) scorbutic diet (Cooper Nutrition). Chronic hypovitaminosis C was induced in 18 of the animals by the daily p.o. administration of 0.5 mg. L-ascorbic acid (Sigma Ltd) in 0.2 ml of 20% sucrose solution. The remaining animals were similarly dosed with 5.0 mg of the vitamin in the same volume of vehicle. This dietary regimen lasted for 5 weeks after which time the animals were weighed, anaesthetized and the bilary tree examined for concrements. Following an overdose of anaesthetic, the cystic duct was ligated prior to cholecystectomy. The gallbladder contents were centrifuged at 5000 rpm for 20 min to separate the bile from gallstones and associated debris,

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