ADP anstatt ATP als Substrat verwendet; die Spaltungsrate betrug dabei rund 30% der in Gegenwart von ATP.

Die ATPase-Aktivität der Mitochondrien, im Saccharose-Dichtegradienten aus der Endfraktion gewonnen, betrug in 2 Versuchen 10,8 ± 1,2 nmol Pi/mg Protein/min; sie war ohne NEM, in Gegenwart von $1 \times 10^{-4} M$ NEM und bei NEM-Zugabe nach ATP-Vorinkubation nicht signifikant verschieden.

Diskussion. Die beschriebene Präparation liefert eine Endfraktion, deren pressorische Aktivität, nach BOUCHER ermittelt und auf Eiweiss bezogen, rund 12mal grösser ist als die pressorische Aktivität des Ausgangshomogenats. Da die reninhaltigen Granula als solche in der Endfraktion ungleich stärker angereichert sind als die pressorische Aktivität, ist anzunehmen, dass während der Präparation Reninverluste auftreten.

Etwa 35% der gefundenen ATPase-Aktivität waren durch NEM hemmbar. Diese NEM-Hemmung konnte durch Vorinkubation mit ATP aufgehoben werden. Solche Eigenschaften sind für Transport-ATPasen charakteristisch[10,11].

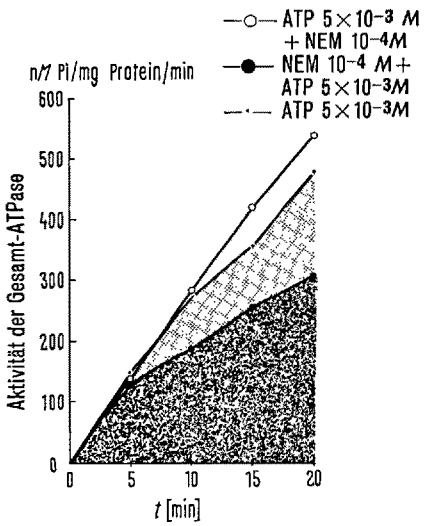

Fig. 2. Zeitverlauf der Wirkung von NEM auf die ATPase-Aktivität der Präparation und die Schutzwirkung von ATP. ATP, Gesamtaktivität; NEM + ATP, 5 min Vorinkubation mit NEM; ATP + NEM, 5 min Vorinkubation mit ATP. Spaltungszeit 20 min.

Für die Annahme, dass die NEM-sensitive ATPase-Aktivität eine Eigenschaft der Reningranula und nicht anderer Bestandteile der Endfraktion ist, sprechen folgende Argumente. 1. Die im vorliegenden beschriebene, vermutlich Reningranula-ständige Transport-ATPase ist im Gegensatz zu mikrosomalen oder Zellmembranständigen Transport-ATPasen nicht Ouabain-empfindlich[12]. 2. Die mitochondriale ATPase ist nicht NEM-empfindlich[13]. Dies trifft nach Kontrollversuchen auch für Mitochondrien zu, die aus unseren Präparaten über einen Saccharose-Gradienten gewonnen worden waren.

Die NEM-insensitive ATPase-Aktivität unserer Endfraktion könnte ausser in Mitochondrien auch in den Reningranula lokalisiert sein. Tatsächlich wurden am Nierenschnitt histochemisch saure Phosphatasen in den Granula des juxtaglomerulären Apparates nachgewiesen[14]. Schliesslich weist auch die renale Lysosomenfraktion bei pH 5 β-Glycerophosphat- und ATP-spaltende Aktivität auf[15]. Im Gegensatz zu den lysosomalen Phosphatasen stieg die ATP-spaltende Aktivität unserer Präparation jedoch bis zum Bereich von pH 8 an.

Summary. In a preparation of renin-containing granules from pig kidneys a NEM-sensitive ouabain-insensitive transport ATPase-activity is described.

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The Effect of Insulin on the Glycolysis and Glycogen Content of Beating Rat Heart Cells in the Primary Culture

The fact that insulin accelerates glucose uptake in skeletal and cardiac muscle is generally accepted[1]. We studied the effect of insulin at the primary culture prepared from chicken embryo heart and newborn rats[2-4]. The beating rat heart cells were prepared according to a modification of the method used by HARARY and FARLEY[5].

Since the culture contains several types of cells, the proportion of which changes with the age of culture[6], we confined ourselves to only a short time for the 3 days in culture. The cultures were incubated with semisynthetic medium[7] without (control group) and with insulin (10 mU insulin per ml medium) 20 h after isolation of the cells. After an incubation period of 24 h or 4 h the glucose uptake, the lactate production and lactate C-14-production from C-14-glucose were determined[8-10]. The pathway glucose to lactate was very active. The large percentage of glucose C-14-uptake appeared as lactate-C-14 (62.8 ± 3.26%, $n = 26$). Insulin increased the lactate-C-14-production from U-C-14-glucose (Table).

Incorporation of radioactivity from U-C-14 labelled glucose into lactate within 4 h

	dpm × 10^4/µmol of produced lactate	
	Control	+ Insulin
$\bar{x}$	4.85	5.83
$s_{\bar{x}}$	0.212	0.166
n	8	10
	$p < 1\%$	

After 24 h of incubation we determined the total cell protein with LOWRY[11] reagent and the glycogen content with anthron reagent[12]. For the determination of glycogen, the cells were heated with 30% KOH for 30 min and precipitated with ethanol and Na_2SO_4 solution as coprecipitant[13]. Insulin enhanced the glycogen content per 100 µg total cell protein (Figure). The difference between control and the culture treated with insulin is significant ($p < 1\%$).

Studies carried out on the beating rat heart cells in primary culture indicated that the cells isolated with fractionated trypsination are suitable for the investigation of the effect of insulin in the muscle cells.

Zusammenfassung. An pulsierenden Rattenherzzellen in der Primärkultur wurde die Insulinwirkung auf die Glykolyse und den Glykogengehalt untersucht.

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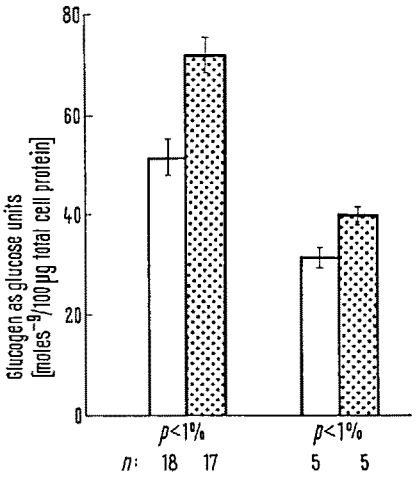

Glycogen content of beating rat heart cells 3 days in culture. Open bars, control; dotted bars, insulin 10 mU/ml medium. $\bar{x} \pm s_{\bar{x}}$.

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The Influence of Fibrinogen Degradation Products on the Electrobiological Activity of the Rat Heart

From earlier publications we know that the pharmacological properties of fibrinogen degradation products (FDP), that is, the potentiation of the smooth muscle contraction caused by the amines or polypeptides, result from the FDP action upon the cell membrane[1-4]. It was found in experiments on the isolated rat uterus that FDP in small doses causes an increase in the amplitude and the frequency of the action potentials, and in higher doses lowers the resting potentials, and at the same time the amplitude and the frequency of the action potentials decrease until they finally disappear altogether[5-7]. In this paper we will present the influence of FDP on the electrobiological phenomena of the rat heart.

Methods. In our experiments in vitro the right auricle of the isolated heart of a Wistar rat was used. 2 methods of preparation of the specimens for the electrobiological investigation were employed.

1. The right auricle was placed in oxygenated Tyrode's solution at 37 °C immediately after decapitation of the animal and excision of the heart and 10–15 min after we began the electrobiological measurements. In these specimens, each time the microelectrode was introduced into the heart cell it was possible to register the resting potential and the action potentials, the amplitude and frequency of which were regular.

2. The auricle was incubated for about 2 h in oxygenated Tyrode's solution at 20 °C. In such a preparation, an introduction of the microelectrode, only resting potentials were registered in the majority of the cases.

The electrobiological investigations were carried out in a 25 ml water bath at 37 °C. Glass (Pyrex) microelectrodes of about 0.5 µm in diameter were used. These were filled with 3 *M* KCl solution. A silver, zero electrode, was placed near the tissue investigated. The registering microelectrode, which was connected by a silver wire to an electrometric lamp via a D.C. amplifier, fed the electrical potentials into the oscilloscope (OK7-2S).

Partially purified products of the proteolytic degradation of fibrinogen (Kekwick) digested by plasmin were used in the experiments[2,6]. The concentration of FDP was expressed in µg of tyrosine calculated per 1 ml of Tyrode's solution in the water bath.

Results. Fibrinogen degradation products in a concentration of 2.5 µg tyrosine acting on the muscle of the right auricle, which shows regular spontaneous electrobiological activity, cause a transient increase in the frequency of the action potentials (Figure 1a). When

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