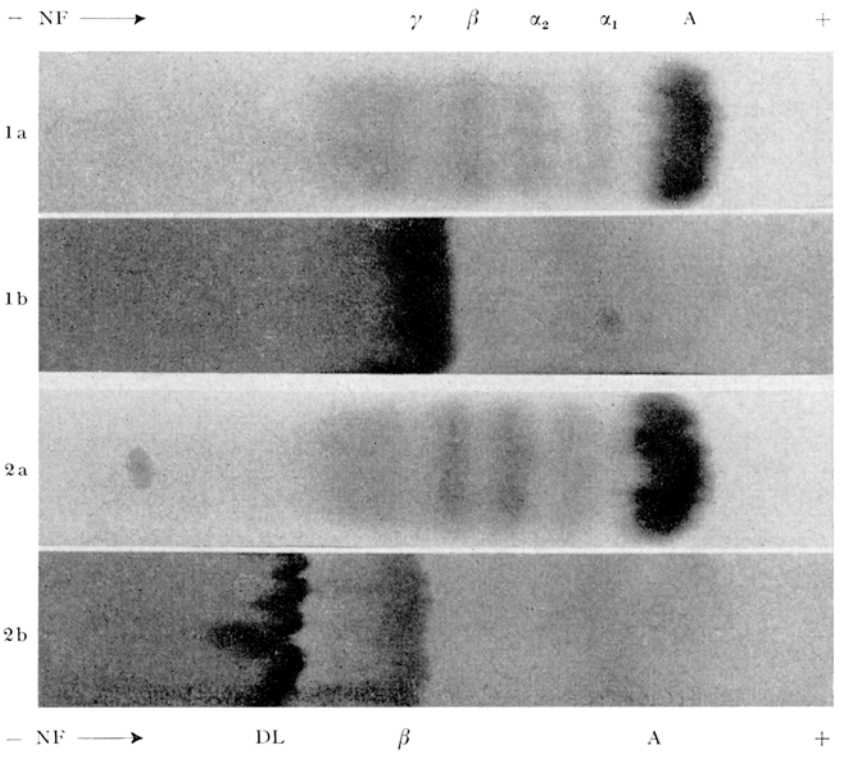
volumes of the colloidal solution of dextran was added to one volume of serum. The turbidity of serum was estimated at 590 mμ on the Zeiss Elko II photometer. The filter paper strip electrophoresis was performed in veronal Na-acetate and in phosphate buffer at pH 8.6 in a potential gradient of 3–4 V/cm for 18 h. The filter paper used was Whatman No. 1. The lipid patterns were stained with Sudanblack B and Oil Red 0, the protein patterns with Amidoblack 10 B and Bromphenolblue.



The effect of dextran in colloidal solution on lipids and proteins as recorded by paper strip electrophoresis.

1. Normal serum. 2. The action of dextran on the same serum. NF = neutral fats; DL = the dextran-lipid fraction. a = protein staining. b = lipid staining.

Results. In all experiments performed *in vivo* and *in vitro* (10 cases), dextran in colloidal solution exerted no change in turbidity of fasting and lipaemic human sera. On the contrary, in all the experiments performed, *in vivo* and *in vitro* with lipaemic or fasting sera, dextran in colloidal solution changes the lipid patterns in paper strip electrophoresis. A new lipid fraction appears with a slower electrophoretic mobility than any known protein or lipid fraction. It appears between the starting point and the γ-globulins. We suggest calling this new fraction the dextran-lipid fraction (DL-fraction). The Figure represents a typical result of the experiments. The appearance of the DL-fraction obviously diminishes the intensity of the lipid pattern migrating with the mobility of the β-globulins. The protein patterns seem not to be altered by the action of dextran. It seems to us that the DL-fraction contains no proteins but an appreciable amount of dextran. Thus we assume that this fraction represents a dextran-lipid complex. Further work on the subject is in progress and will soon be published in detail elsewhere.

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Zusammenfassung

Eine 6%ige kolloidale Dextranlösung, *in vivo* infundiert oder dem Serum *in vitro* zugesetzt ändert dessen Lipidogramm. Zwischen Startpunkt und γ-Globulinen entsteht eine neue mit DL bezeichnete Fettfraktion. Im Verlauf der Experimente unterblieb eine Trübung des Serums und Änderung des Elektrophorese-Proteinogramms.

Serotonin Inhibition of Liver Mitochondria Swelling *in vitro*

In recent researches on mitochondrial morphology, it has been found that many substances possess the ability of inhibiting the swelling which occurs spontaneously when the particles are suspended in isotonic sucrose: such substances are for example ATP (adenosine triphosphate), Mg^{++}, versene, and K^{+}[1–3]. Other substances, such as phosphates or succinate, increase or accelerate this process of water exchange between the medium and mitochondria in relation to their metabolic activity[3–5]. Recently some neurohumoral amines have been considered for their action on mitochondrial morphology: histamine, adrenaline, and acetyl choline were found to be completely inactive *in vitro*[6]. In this note the effect of another neurohumoral amine, 5-hydroxytryptamine (HT), on spontaneous swelling of rat liver mitochondria *in vitro* has been studied. This neurohumoral amine is contained in appreciable amounts in blood platelets; and since these do not contain enzymes capable of metabolising it, and since substances have been found which influence the binding or release of HT from blood platelets, such as reserpine[7], it would seem that they merely have a function of transport with respect to HT. Walaszek and Abood, studying brain mitochondria, found that they contain a considerable

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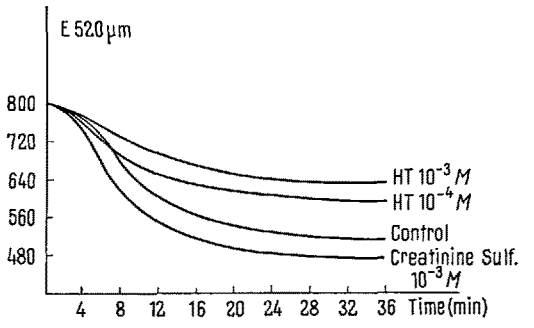

Inhibition by Serotonin on Mitochondrial Spontaneous Swelling as Estimated by the Optical Method, and Swelling Effect of Creatinine-Sulfate.

Percentage Decrease (D%) of Optical Density on Spontaneous Swelling of Rat Liver Mitochondria in 35 min

	No. of experiments	D%
Control	15	29,4 ± 6,4
Mitochondria with serotonin 10^{-3} M	10	20,8 ± 4,5
Mitochondria with serotonin 10^{-4} M	7	22,3 ± 5,1
Mitochondria with creatinine-sulfate 10^{-3} M	3	32,7 ± 5

amount of HT. In addition they observed that reserpine has an effect on the serotonin of these mitochondria which is parallel to that demonstrated on blood platelets[8]: it therefore seemed interesting to see whether this neurohumoral amine influenced mitochondria morphology.

Mitochondria were prepared from liver of albino rat, Wistar strain (150–200 g) body weight) in 0.25 *M* sucrose, containing 0.02 *M* Tris-chloride, pH 7.4 at 4°C, by the method of Schneider[9]. 1 ml of the final concentrated suspension of mitochondria was equivalent to 0.25 g of fresh liver. Changes in extinction at 520 mµ read in a Beckmann model DU spectrophotometer, were taken as measure of swelling as described by Cleland[1]. Each test tube contained 3 ml of 0.25 *M* sucrose, buffered with 0.02 *M* Tris-chloride, pH 7.4: HT was added to the sample examined. Then 0.3 ml of the mitochondria suspension were quickly added, the tube was shaken to mix its contents and the optical density was determined. Usually the first reading was taken within 20–30 sec after mixing and then at given times up to 35 min. The zero time or initial optical density was obtained by extrapolation. The whole experiment was made within 75 min from the killing of the animal.

The 5-hydroxytryptamine-creatinine sulfate (it is the only salt available) used was supplied by Sigma and Roche Laboratories. The results are given in the Table: a significant difference was observed in the % decrease of optical density in samples containing HT; the Figure gives the effect of serotonin on spontaneous mitochondrial swelling at two different concentrations. We also verified the effect of creatinine-sulfate on mitochondria: it was found that it has a swelling effect and is therefore an antagonist to HT.

Consequently it appears that serotonin has a protective action on mitochondria swelling *in vitro*. Further studies are being carried out in order to see if there is a correlation between ATPase activity and protective action of serotonin. This is supported by the fact that the concentration of HT in blood platelets depends on the ATP present[10] (according to Born[11], serotonin would actually be bound to ATP as a complex) and that inhibitors of HT binding by mitochondria such as reserpine, dibenamine, and phenylether, inhibit oxidative phosphorylation[9].

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Riassunto

È stato studiato l'effetto della serotonina sul rigonfiamento dei mitocondri *in vitro:* si è trovato che la serotonina protegge i mitocondri dal rigonfiamento spontaneo che si ha quando vengono sospesi in soluzione isotonica di saccarosio.

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The Kinetic of Induction of Plaque Formation in Cell Monolayers by Ribonucleic Acid from Poliovirus

We have published previously[1] a test system for quantitative studies on the infectivity of isolated ribonucleic acid (RNA) from poliovirus. The method was developed to provide a tool for physico-chemical analysis of biological active RNA, in a manner comparable to the analysis of desoxyribonucleic acid (DNA) carried out with the aid of studies on transforming DNA. Other investigators have already applied this method for further characterization of infectious RNA[2,3]. It was of interest to learn more about the basic mechanism of the RNA test system. We present here results on the kinetic of plaque induction in cell monolayers by RNA.

The methods used for preparation and assay of RNA are essentially the same as those described previously[1]. Poliovirus type I strain Mahoney is used for isolation of RNA. The virus suspensions are obtained partially purified from the Cutter Laboratories. Highly purified poliovirus suspensions are prepared in our own laboratory according to the method of Levintow and Darnell[4]. The experimental results obtained are independent of the virus source.

The solutions for washing the cell monolayers and the RNA solution are incubated in a water bath at the stated temperatures. The cell monolayers are kept at these temperatures for 20 to 30 min before seeding with the RNA.

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