Time of ultrasonic treatment	Enzyme activity in Willstätters units	pH values of solution
0 min	9.9×10^{-4}	5.50
5 min	8.5×10^{-4}	5.50
10 min	7.0×10^{-4}	5.20
15 min	5.6×10^{-4}	5.05
20 min	4.8×10^{-4}	4.90
30 min	2.7×10^{-4}	4.70

The results obtained show that the activity of 0.5% diastase solutions and the pH values have been decreasing gradually but regularly.

These investigations are of significance for application of ultrasound in practical medicine as well. The mechanism of the action of ultrasound being still insufficiently explained, it is necessary to be careful when applying it for therapeutic purposes. Just for that reason, the investigation of its action on substances contained in human body is of great importance, since it offers the possibility of avoiding mistakes and accidents when applying ultrasound for therapy. We have investigated the action of ultrasound on diastase solution (0.5% B. D. H. make) and in each experiment we have used 10 ml of it.

The ultrasound of frequency 3 Mc/s and intensity of 1.5 W/sq.cm. has been obtained from a Yugoslav make of generator. The solutions have been placed in a flask, the bottom of the flask being made of thin gum. The samples were exposed for the period of 0, 5, 10, 20, and 30 min respectively. During the exposure the solutions have been cooled in tap water in such a way that the temperature has never been allowed to rise above 30°C. The activities of the solutions were determined by WILLSTÄTTER'S method[9] 5 h after the treatment. The pH values were measured immediately after exposure (Table).

V. STEFANOVIĆ, A. DJUKANOVIĆ, K. VELAŠEVIĆ, and D. ŽIVANOVIĆ

Institute of Tuberculosis of the P. R. of Serbia, Belgrade, Institute of Inorganic and Analytical Chemistry, Pharmaceutical Faculty, Belgrade, and Institute of Biology, Veterinary Faculty, Belgrade (Yugoslavia), March 11, 1960.

Zusammenfassung

Die Ultraschallwirkung auf Diastase wurde an deren Aktivitäts- und pH-Änderung untersucht. Verschieden lang beschallte, 0.5%ige wässerige Diastaselösungen ergaben stufenweises und regelmässiges Absinken der Aktivitäten und pH-Werte.

The Inhibitory Effect of 6-Azauracil Riboside on the Multiplication of Vaccinia Virus

6-Azauracil, and particularly its riboside, are known to inhibit the growth of a number of microorganisms[1,2] and experimental tumours[3-5]. We now wish to report tests on the anti-virus activity of the two compounds.

In our screening test (diffusion of the tested substance through agar using Dulbecco's plaque method for virus giving a positive cytopathogenic effect) 6-azauracil proved inactive but its riboside exhibited notable activity on the multiplication of vaccinia virus compared with benzimidazole (Figures 1–3).

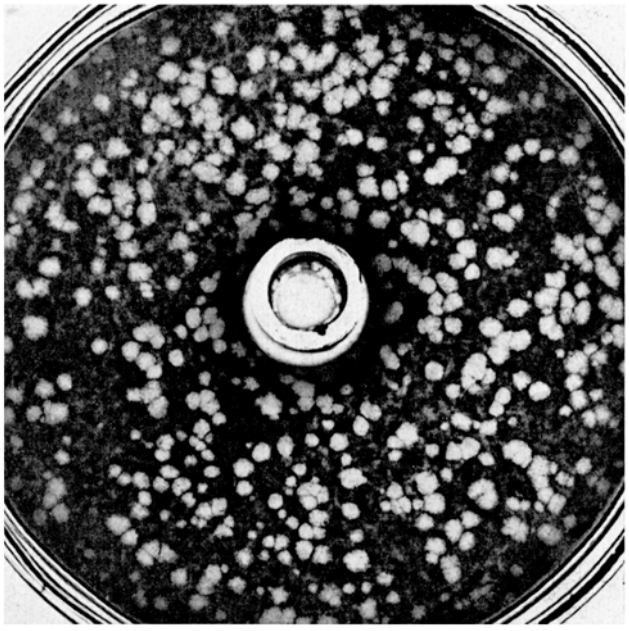

Fig. 1. 6-Azauracil (4.4×10^{-1} *M*, incompletely dissolved). No zone of inhibition or toxicity.

Fig. 2. Benzimidazole (8.5×10^{-2} *M*, Fluka, Switzerland). Zone of toxicity around the cylinder and further away narrow zone of inhibition.

[1] F. Šorm and F. Škoda, Chem. listy *50*, 827 (1956); Coll. Czechoslov. chem. Comm. *21*, 487 (1956).

[2] R. E. Handschumacher and A. D. Welch, Cancer Res. *16*, 965 (1956).

[3] F. Sorm, A. Jakubovič, and L. Šlechta, Exper. *12*, 271 (1956).

[4] T. M. Hakala, L. W. Law, and A. D. Welch, Proc. Amer. Ass. Cancer Res. *2*, 113 (1956).

[5] F. Šorm and H. Keilová, Exper. *14*, 215 (1958).

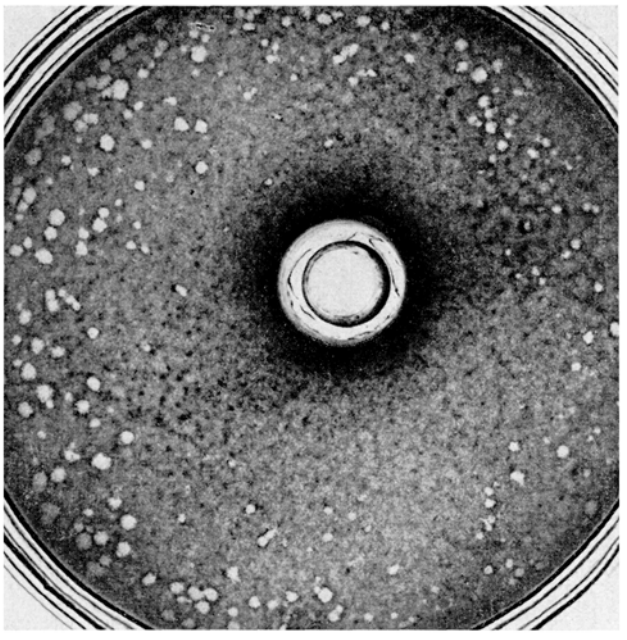

Fig. 3. 6-Azauracil riboside[6] (2.0 *M*). Large zone of inhibition without zone of toxicity.
The cultures were seeded with approximately 500 plaque-forming units of vaccinia virus; the cylinders were charged with 0.05 ml of solution of the substance tested; dishes of 4 cm diameter were employed.

A quantitative evaluation of the effect of 6-azauracil riboside on the multiplication of vaccinia virus was performed, using the method of Overman and Tamm[7] with the modification that the ineffectivity test was carried out by Dulbecco's plaque method on chick fibroplast cultures. In the toxicity tests, the method and criteria of Tamm[8] were adopted. 6-Azauracil riboside has very low toxicity: only concentrations as high as 1.75×10^{-1} *M* bring about changes in the curling up of a piece of chorioallantoic membrane characterised by Tamm as 2 +. On the other hand, a 1.2×10^{-2} *M* concentration of 6-azauracil riboside causes a 75% inhibition of the multiplication of vaccinia virus. Its selectivity (ratio of toxicity to virus inhibiting concentration) is thus about 14, i. e. sufficient to permit its use in amounts not toxic for the animal host.

6-Azauracil riboside has no direct inactivating effect on vaccinia virus, as shown by incubation of the virus with 4×10^{-2} *M* 6-azauracil riboside for 3 h at 35°C (Table).

6-Azauracil riboside was found to have no effect on influenza virus type A strain PR8, when tested by the method of Tamm *et al.*[9], and against Newcastle disease virus (NDV) and Eastern equine encephalomyelitis virus (EEE), when tested by the plaque method.

Number of Plate Forming Units (PFU)/ml in Presence of 6-Azauracil Riboside

h	Medium + 6-azauracil riboside[a]	Blank
0	2.8×10^5	2.8×10^5
3	1.5×10^5	2.4×10^5

[a] The concentration of 6-azauracil riboside was 4×10^{-2} *M* which corresponds to 99% inhibition in the membrane culture test.

B. Rada, D. Blaškovič, F. Šorm, and J. Škoda

Institute of Virology, Czechoslovak Academy of Science, Bratislava and Institute of Organic Chemistry and Biochemistry, Czechoslovak Academy of Science, Prague, April 29, 1960.

Zusammenfassung

Die Hemmwirkung von 6-Azauracilribosid auf die Vermehrung des Vaccina-Virus in Gewebekulturen wurde festgestellt. Die Substanz blieb gegen andere Viren (Influenza A, NDV, EEE) wirkungslos.

[6] J. Škoda, V. F. Hess, and F. Šorm, Exper. *13*, 150 (1957); Coll. Czechoslov. chem. Comm. *22*, 1330 (1957).
[7] J. R. Overman and I. Tamm, Virology *3*, 173 (1957).
[8] I. Tamm, J. Bacteriol. *72*, 42 (1956).
[9] I. Tamm, K. Folkers, and F. L. Horsfall, Jr., J. exp. Med. *98*, 229 (1953).

A Paper Strip Electrophoretic Examination of the Action of Dextran in Colloidal Solution on Human Serum

Sulphated polysaccharides applied *in vivo*, as heparin and dextran sulphate, produce a clearing effect on human and animal lipaemic serum[1–7], and an accelerating effect on the electrophoretic mobility of the serum lipids migrating with the β-globulins and albumins[1,5,8,9].

These sulphated polyanions interact with serum *in vitro* forming an insoluble complex with the lipids migrating electrophoretically with the β-globulins[10–13]. This insoluble complex also contains proteins.

In the present paper, a preliminary investigation of a possible effect of dextran in colloidal solution on turbidity of normal and lipaemic human sera is tried. A possible action of dextran in colloidal solution on electrophoretic mobilities of serum lipids was also examined. The experiments were performed with human sera by applying dextran *in vivo* and *in vitro*.

Materials and Methods. The dextran used in infusion *in vivo* and experiments *in vitro* was 'Macrodex' from Pharmacia Uppsala Sweden in a 6% colloidal solution. The infusion was given to healthy males and females during 2 h at a rate of 210 mg/kg/h. In the experiments *in vitro* 0.30

[1] S. Gerö, M. Baumann, M. Jakab, S. Rohny, and J. Gacs, Z. ges. exp. Med. *129*, 335 (1957).
[2] W. Schuler and G. Müller, Schweiz. med. Wschr. *87*, 787 (1957).
[3] H. Engelberg and D. Rodensky, Circulation *6*, 266 (1958).
[4] N. Zöllner and H. D. Frings, Z. ges. exp. Med. *122*, 275 (1956).
[5] A. Benaš, M. Keler-Bačoka, and Z. Pučar, in preparation.
[6] J. L. Beaumont, B. Beaumont, B. Boumard, and J. Lenègre, Bull. Soc. méd. Hôp. Paris *74*, 63 (1958).
[7] W. D. Brown, J. exp. Med. *37*, 75 (1952).
[8] S. M. Jeavons and C. R. Ricketts, J. clin. Pathol., London *9*, 225 (1956).
[9] P. Bernfeld, V. M. Donahue, and M. E. Berkovitz, J. biol. Chem. *226*, 51 (1957).
[10] A. Scanu, L. A. Lewis, and J. H. Pag, J. Lab. clin. Med. *51*, 325 (1958).
[11] J. L. Oncley, K. W. Walton, and D. G. Cornwell, J. Amer. chem. Soc. *79*, 4666 (1957).
[12] H. N. Antoniades, J. L. Tullis, L. H. Sargeant, R. B. Pennell, and J. L. Oncley, J. Lab. clin. Med. *51*, 630 (1958).
[13] M. Burstein and J. Samaille, Clin. chim. Acta *3*, 320 (1958).