the animal. It may be that the virus is so rapidly changed in the animal body that it has no chance to react with the tumor cell, or the known inhibitors present in all animal tissues may interfere. Perhaps much more virus is needed to produce the effect *in vivo* than *in vitro*.

The results reported are in complete agreement with those reported by Kausche, Landschutz and Sauthoff, who had observed the same phenomenon with the PR8 strain of influenza and Ehrlich tumor cell suspensions. Their explanation of the phenomenon, *i.e.* growth of the virus in the tumor cells, are at variance with ours since we have been unable to demonstrate any increase in viral titer in either the ascitic fluid or in the solid tumor following direct inoculations. It appears to us that the tumor inhibiting effect is more likely to be due to an adsorption of virus on the cell surface which for some unexplained reason is capable of retarding tumor growth when these mixtures are inoculated either intraperitoneally or subcutaneously into animals. The fact that these same mixtures on further incubation or on a change of temperature from 4° to 25° are capable of growing when inoculated, seems to indicate a surface phenomenon.

Summary. 1. When NDV and Ehrlich were mixed at 4°, 25° and 37° and inoculated immediately, a much smaller percentage of tumors grew than in the NAF control mixtures. When the same mixtures were held at 25° or transferred from 4° to 25° for one to 3 hours, inoculation resulted in tumor growth. 2. When virus and tumor were mixed *in vivo* no tumor inhibitory effect was noted unless the virus was given intraperitoneally first and the tumor suspensions by the same route immediately thereafter. The course of development of ascites in mice was not affected by daily inoculations of NDV. 3. No evidence could be found for multiplication of the virus in the tumor cells either in the animal or tissue culture.

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Dextran as a Source of Liver Glycogen and Blood Reducing Substance.* (19922)

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In earlier experiments in this laboratory it has been observed that dextran, fed to human subjects, is not recovered in the stools. Engstrom and Aberg(1) have suggested that some of the dextran administered intravenously might be excreted into the gastrointestinal tract where it could be destroyed by bacterial action. Our own observations on dextran incubated with stools suggested that dextran disappears only very slowly, but Hehre(2) has since shown that there are large numbers of anaerobic bacteria present in the intestinal flora, and that it is these, rather than the aerobes, that are capable of splitting dextran. Although ingested dextran may be in part degraded and consumed by the intestinal bacteria, it still seemed to us of interest to learn more about the disappearance of dextran fed by mouth. The following experiments show that dextran feeding leads to a sustained increase in blood sugar and a rise in liver glycogen in the fasted rat, and brings about an

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TABLE I. Blood Reducing Substance Following Administration of 5 ml of 18% Dextran by Stomach Tube to 24-Hr Fasted Rats.

Time in hr	0	1	2	3	4
No. of rats	6	4	4	2	2
Reducing substance (mg %)	80 ± 7.9	113 ± 3.5	100 ± 8.2	96.5 ± 2	95.5 ± 2

increase in the blood sugar of human subjects.

Experimental. Male albino rats of the Sprague-Dawley strain, weighing 250-350 g were fasted for 24 hours. A group of 10 control animals were killed and both fractions of liver glycogen were determined by the method of Bloom, Lewis and Schumpert(3). Glycogen was determined (as glucose equivalent) by means of the anthrone reaction. The fasted experimental animals were given 5 ml of 18% dextran in 0.9% saline by stomach tube. An initial blood sample was taken from the cut end of the tail and additional samples were taken at intervals after feeding. Copper tungstate filtrates were prepared and reducing substances were determined by the method of Nelson(4). In a series of four rats, both total and fermentable reducing substances were determined. Two or four hours after feeding, the rats were anesthetized with Nembutal and the liver was removed and immediately frozen and powdered. Trichloroacetic acid-extractable and total glycogen was determined on aliquots of the well-mixed liver powder. In order to determine whether glycogen or dextran might be present, other samples were allowed to stand at room temperature for 1.5 to 2 hours in order that glycogenolysis might occur. Dextran was added to parallel samples in order to determine whether dextranolysis could occur in the liver preparation.

To two human subjects, fasted for 12 hours, 100 ml of 20% dextran were given orally and blood sugar was determined at 0, 0.5, 1 and 2 hours thereafter. Two other patients were fasted 12 hours, 200 ml of 20% was given orally and blood sugars determined at the described intervals.

Results. The data of Table I show that the blood reducing substance in dextran-fed rats increases significantly in one hour after feeding and is still above the initial level after four hours. In Table II it is seen that the increase in blood reducing substance (of about the same extent but of shorter duration after feeding half the amount of dextran given to the first series of animals) is entirely fermentable. Table III presents the data on two human subjects given 20 g of dextran by mouth. The increases in blood sugar in 0.5 to 1 hour are of the same order as those seen in the rats.

In Table IV it is seen that there is a considerable increase in liver glycogen four hours after feeding dextran to previously fasted rats, and that allowing the liver samples to stand

TABLE II. Total and Non-Fermentable Blood Reducing Substance in 24-Hr Fasted Rats Given 5 ml of 9% Dextran by Stomach Tube.

Wt, g	Time after dextran	Blood reducing substance (mg %) Total	Non-fermentable
285	Control	99.2	0
	30 min.	116.2	0
	2 hr	86.1	0
	4 "	73.2	0
300	Control	80.1	0
	30 min.	121.7	0
	2 hr	74.8	0
	4 "	63.6	.25
265	Control	87.4	0
	30 min.	104.6	0
	2 hr	81.8	2.5
	4 "	67.7	.5
250	Control	65.5	12.7
	30 min.	97.6	0
	2 hr	81.8	4.3
	4 "	68.5	1.3

TABLE III. Blood Reducing Substance Following Administration of 100 ml of 20% Dextran to 12-Hr Fasted Human Subjects. (Values for reducing substance in mg %.)

	Subject I	II	III	VI
Control	83	100	90.5	118
½ hr	107	110	103	161
1 "	99	126	101	137
2 "	89	112	104	128

TABLE IV. Liver Glycogen Concentration in Fasting and Dextran Fed Rats.

	Alkali soluble, mg/100 g	TAA soluble, mg/100 g
10 fasting rats	130 ± 13*	31 ± 8
14 fasted rats 4 hr after dextran feeding		
Immediate analysis	751 ± 106	469 ± 66
Aliquot at room temp. 2 hr	71 ± 13	10 ± 2

* Stand. error.

TABLE V. Recovery of Added Dextran from Livers Undergoing Glycogenolysis.

No. of livers analyzed	Dextran added, mg/g	Dextran recovered, mg/g	
		Alkali soluble	TAA soluble
6	644	517 ± 31*	610 ± 8
8	736	720 ± 22	683 ± 20

* Stand. error.

The dextran was added to each of two samples, one of which was digested with alkali, the other of which was extracted with trichloroacetic acid. Time of incubation at room temp. was 2 hr.

for 2 hours at room temperature results in the disappearance of most of the glycogen. Dextran added to aliquots of the liver tissue and determined either as alkali-soluble or trichloroacetic acid-soluble anthrone-reacting substance, is recovered without substantial loss, so that no significant dextranolysis seems to occur under these conditions in liver tissue (Table V).

Discussion. The data show that in both rat and man the oral administration of dextran leads to a significant and sustained increase in blood reducing substance, most of which is fermentable. In the rat, this increase in blood sugar is accompanied by a significant increase in liver glycogen 4 hours after feeding. Dextran is therefore capable of being broken down in the intestine to products which yield glucose and glycogen in the animal. The early increase in blood sugar in both rat and man indicates that the intestinal breakdown of dextran may be a relatively rapid process, and it suggests that this may not be ascribable merely to bacterial action but more probably to an action of an enzyme or enzymes of the intestinal tract. Experiments now under way in this laboratory indicate that the latter possibility may be realized: suspensions of rat duodenal mucosa have been found to liberate glucose from dextran at rates which can account satisfactorily for the increases in blood sugar seen after feeding.

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Plasma Volume Determination in the Hypovolemic State Produced by Intraperitoneal Injection of a Non-Electrolyte Solution. (19923)

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It was noted in previous work(1), that following the subcutaneous injection of non-electrolyte containing fluids there were certain discrepancies in plasma volumes as determined by the RIHSA* method. These were manifested by failure to obtain reduction in plasma volume commensurate with the rise in hematocrit, and clinical signs of hypotension and dehydration. One of the possible factors considered was an abnormal rate of disappearance of the radioactive albumin in this pathophysiologic state. It was thought to be worth-

* Radioactive Iodinated Human Serum Albumin.