The antigenic relationships shown in the present and previous(1,2) studies are in general agreement with the results of recent investigations by other workers, insofar as comparisons can be made. Thus, van der Veen and Mulder(22) found the A viruses isolated between 1934 and 1943 to be distinct from the A prime group (1946-1949) with few exceptions, and the WS agents, which were clearly separated from the A prime strains, were reasonably distinct from such A type viruses as PR8. Andrewes and Isaacs(16) recently reported the occurrence of 2 antigenic types of influenza A prime virus among 74 strains isolated during 1950-51; these were differentiated by the agglutination-inhibition test with ferret antisera treated to destroy normal inhibitor. Strains designated S reacted in general to very low titer with all A prime ferret sera, even homologous ones, and the L viruses reacted to high titer with homologous ferret sera and much less with other A prime sera. A few strains with intermediate properties were designated SL. The 1950-51 strains we examined (only 5 of our viruses could have been the same as those of the above study) did not show such differences; in contrast, our strains were remarkably homogeneous in spite of the high level of strain specificity which chicken antisera exhibit. The marked antigenic difference between Lee virus and most of the influenza B strains isolated since 1945 was likewise shown in a report by Tamm and associates(23). The present studies confirmed the antigenic identity of 1233 and JJ viruses reported by Francis and associates(6).

Summary. A method for designating antigenic components of influenza A, B and C viruses consisting of formulae derived from the results of agglutination-inhibition tests with chicken antisera prepared against a selected group of prototype strains was presented and discussed. Studies to determine the antigenic patterns of some recently isolated influenza A viruses by the agglutination-inhibition technic revealed that the A prime strains isolated in 1950-51 formed a remarkably homogeneous group resembling the 1950 FW-1-50 (Cuppett) agent. The Benson virus, which was an exception, was indistinguishable from the 1947 FM1 strain. The results of the antigenic analyses of some WS- and PR8-like viruses isolated since 1947 (during which time the A prime form of influenza A was predominant) were also included and their significance discussed.

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Effect of Hypoglycemia on Rats with Intrasplenic Adrenal Transplants.* (19024)

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Previous reports(1-3) have shown that auto- and homotransplants of the adrenal gland to the spleen of adrenalectomized rats, maintained life, maturation and gonadal function in the recipients. It was suggested that the liver did not destroy the adrenal

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TABLE I. Effect of Intraperitoneal Injection of 250/kg Alloxan on Rats, with Intrasplenic Adrenal Transplants (Group A), Intrarenal Adrenal Transplants (Group B), Bilateral Adrenalectomy (Group C), and Unilateral Adrenalectomy (Group D).

Rat No.	Fasting* blood sugar	Diabetes: Time after alloxan (hr)	Diabetes: Blood sugar	Time of death: Hr	Time of death: Blood sugar
Group A					
1	55	24	153	36–48	
2	51	6	160	36–48	
3	51	24	134	36–48	
4	55	24	294	36–48	
5	54	24	343	36–48	
Group B					
1	55	24	426	50 (sacrificed)	
2†	65	6	162	24 (killed by heart puncture)	
3†	55	‡	‡	36–48	
4†	49	6	252	36–48	
5	49	6	130	36–48	
Group C					
1	41			2	3
2	46			2	3
3	44			2	0
4	34			2	7
5	49			2	5
Group D					
1	45	24	290	48–72	
2	55	24	176	24 (killed by heart puncture)	
3	41	24	244	Alive at 96 hr	
4	43	24	280	48–72	
5	32	24	200	48–72	

* All animals were fasted 16 hr before inj of alloxan. Blood sugar values are expressed in mg %.

† At 24 hr Rats 2, 3, and 4 of Group B had blood sugar values of 31, 29, and 17 mg respectively.

‡ Readings not obtainable.

secretions responsible for the maintenance of these activities.

The purpose of the present investigation is to present evidence that the adrenal secretions which influence sugar metabolism, are likewise not demonstrably affected by passage through the portal circulation.

Method. Twenty mature Long-Evans rats of approximately the same age were divided into 4 equal groups. Group A had been adrenalectomized and had one adrenal autotransplanted to the spleen about 5 months before the experiment. Group B had been adrenalectomized and had one adrenal autotransplanted to the kidney approximately 4 months before the experiment. Group C was adrenalectomized 3 days before the experiment and maintained on saline instead of tap water. Group D had only one adrenal removed 3 days before the experiment. All animals were maintained on a regular diet except Group C as indicated, until 16 hours before the experiment, at which time all food was removed. Fasting blood sugars† were taken before the intraperitoneal injection of 250 mg of alloxan per kg of rat. Blood sugar determinations were made at various intervals during the course of the subsequent 48 hours, and autopsies were performed on Groups A and B.

Results. All of the bilaterally adrenalectomized animals (Group C) died within 2 hours after alloxan injection from hypoglycemic convulsions, during which they had exceedingly low blood sugar values (Table I). None of the remaining animals died within 36 hours except 2 that were killed inadvertently by

† Micro method of Kingsley, J. R., and Reinhold, J. J., *J. Lab. and Clin. Med.*, 1949, v34, 713.

heart puncture at the end of 24 hours. Four animals of Group B showed definite diabetic blood sugar levels within 24 hours, but after this time, 3 of the 4 animals changed to hypoglycemic levels. Their actual death was not observed but occurred 12 to 24 hours after these low sugar level values were attained.

The autopsies on the animals of Group A revealed that all but Rat No. 1 of Group A had large adrenal transplants in the spleen which averaged 4 mm in diameter, and there were no adhesions that would permit circumvention of the portal circulation. This animal showed no evidence of a transplant, however, a 2.5 adrenal accessory was found.

The autopsies on the animals of Group B revealed that 4 out of 5 showed large transplants that varied from 3 to 5 mm in diameter. The fifth animal (No. 2) had a smaller transplant measuring 1 x 2 mm, no accessory adrenal tissue was found. On histologic examination the adrenals that were transplanted to the kidney, unlike those in the spleen, showed some clearly defined medullary tissue in addition to the usual cortical cells.

Discussion. When alloxan has been administered to a normal rat, preceding the establishment of the diabetic state, there is an immediate rise in blood sugar followed by severe hypoglycemia. The latter state is presumed to be due to release of insulin from damaged islets of Langerhans(4-6). Kirschbaum *et al.* (7) reported that after the administration of alloxan to adrenalectomized rats, they all showed hypoglycemic convulsions within 6 hours with the dosage employed, whereas convulsions did not appear in a control, intact group. This reaction was not influenced by absence of the adrenal medulla but was dependent on the adrenal cortex.

In the present experiment, alloxan was employed to induce hypoglycemia. The 5 adrenalectomized animals (Group C) died in 2 hours with convulsions from marked hypoglycemia, as expected from Kirschbaum's report. The control Group D, with one adrenal removed and one left *in situ,* behaved identically as the group with an adrenal in the portal circulation (Group A). The second control (Group B) with an adrenal transplanted to the peripheral circulation, *i.e.,* the kidney, also behaved like intact animals. Since none of these animals died from hypoglycemia, as did the adrenalectomized group, it is suggested that the secretions from the adrenal cortex concerned in carbohydrate metabolism that are elaborated by the intrasplenic adrenal transplant, are not destroyed by passage through the liver. However, since the site of action of cortical hormones is unknown, it can not be stated with certainty that the adrenal protective mechanism against hypoglycemia is not mediated in the liver *per se.*

There is indirect evidence that animals bearing a transplanted adrenal gland do not tolerate stress as well as animals with adrenal tissue *in situ.* The animals of Group D definitely survived longer than Groups A and B when subjected to the same stresses of starvation and diabetes.

Summary. The administration of alloxan to the adrenalectomized animals resulted in hypoglycemic convulsions and death in 2 hours. This did not occur in animals with an adrenal transplanted to the portal circulation, to the peripheral circulation, or when one adrenal gland was removed and the other left *in situ.* Thus indirect evidence is presented that the liver does not destroy the secretions of the adrenal cortex that may be concerned with carbohydrate metabolism.

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