Conclusion. There is no significant difference between the percentages of cell types in the anterior lobe of the pituitary glands of 15 sexually mature male rats with diabetic traits and those of controls.

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Specific Neutralization of Cotton Rat Strains of Poliomyelitis Virus.*

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Paralysis has been produced in the eastern cotton rat by injecting combinations of poliomyelitis virus and "enteric toxin filtrate."[1] (Virus strains isolated were: Flexner's M. V., Flexner's Philadelphia, and SK New Haven.) In addition, Armstrong's and Jungeblut's strains have been carried 30 and 20 generations in rats, respectively. Flexner's M.V. rat-adapted strain was found to be specifically neutralized by poliomyelitis antiserums.[2] This paper is a report on neutralization experiments with Flexner's Philadelphia, Armstrong's Lansing and Jungeblut's strains.

The set-up of the experiments was similar to those previously reported.[2] There were 5 groups of animals, 30 in each group. Those in Group A were injected with a mixture of the antiserums used—equal amounts of serum and saline. Group B received 4% virus in saline mixed with an equal amount of serum termed "early horse serum" obtained from horses which were found lacking in virucidal antibodies for poliomyelitis virus.[3] Group C received 4% virus in saline and an equal amount of serum termed "convalescent horse serum" obtained from the same horses a few years later after they had been injected with virus.[3] Group D received 4% virus in saline plus an equal amount of pooled human convalescent poliomyelitis serum, and Group E, 4% virus plus saline to make a 2% suspen-

* Sponsored by The National Foundation for Infantile Paralysis, Inc.

1 Toomey, John A., and Takacs, William S., PROC. SOC. EXP. BIOL. AND MED., 1940, **45**, 364.

2 Toomey, John A., and Takacs, William S., PROC. SOC. EXP. BIOL. AND MED., 1941, **46**, 319.

3 Toomey, John A., *Am. J. Dis. Child.*, 1937, **52**, 1492.

sion. The amounts injected were: 0.06 cc intranasally, 0.06 cc intracerebrally, and 0.5 cc subcutaneously.

In neutralizing experiments, there are often insufficient negative and positive controls. In these experiments, there were as many in the negative and positive control groups as there were in the virus and serum injected.

Results. A. *Flexner's Philadelphia Strain.* All positive controls (Group E) died and only 1 of 30 negative controls (Group A). All animals of Group B, which received virus plus "early horse serum," died. Four animals (13.3%) in Group C, protected with convalescent horse serum" and 5 (16.6%) in Group D, protected with pooled convalescent human serum, died.

Conclusion. Bearing in mind the percentage of experimental error, the number of natural deaths that would occur and those that would follow immediately or within 24 hours after injection, etc., one could conclude that specificity had been demonstrated and that this strain of rat virus was truly an acclimated one from Flexner's Philadelphia virus.

B. *Armstrong's Lansing Strain.* All the positive controls (Group E) died; all negative controls (Group A) lived. All animals injected with "early horse serum" and virus (Group B) died. Two (6.6%) of Group C, the "convalescent horse serum" protected and 4 (13.3%) of Group D, given pooled convalescent human serum, died.

Conclusion. Bearing in mind the fact that 100% of the positive controls died and none of the negative as contrasted with mortality rates of 6.6% and 13.3% in the protected groups, it can be concluded that here also we dealt with a specific type of poliomyelitis virus acclimated to the cotton rat.

C. *Jungeblut's Strain.* Three specimens of mouse brain virus were received from Jungeblut. This strain was adapted to cotton rats and mice by Jungeblut and Sanders from strain SK New Haven, Generation XI, (Trask and Paul). This virus was easily transferred to other mice and to cotton rats. The latter were used for these experiments. In the doses used by us, the animals usually died of an overwhelming disease within 3 or 4 days after injection; in many instances they had respiratory muscle paralysis. If the animals lived longer than 4 days, they usually became paralyzed in the hind quarters, although some also had respiratory involvement.

Our results with virus received 7/18/40 were opposite to those had with other adapted strains (Flexner's M.V., Flexner's Philadelphia or Armstrong's Lansing). The animals in the negative con-

trol Group A lived. Every animal in the other 4 groups died. There was no prolongation of life between the time of the injection and death in any group, which would denote some specific protection.

The same experiment with the second lot of material obtained from Jungeblut 2/23/41 was repeated in 150 rats and the results were the same. A third experiment with a third lot of Jungeblut's material received 4/9/41 injected into a similar number of animals again gave the same results. No evidence nor tendency toward specific neutralization was demonstrated when cotton rats were used as the test animals.

Jungeblut's original adaptation came from Paul and Trask's SK strain (11th transfer generation). We obtained from Trask some of the 13th generation of the same strain. We had difficulty in adapting it to rats. Paralysis would often appear for 3 generations and then the strain would be lost. It was finally combined with "enteric toxin filtrate" and transferred to the eighth generation, but its virulence was nothing like that of the other acclimated strains. The animals injected with our adapted Trask virus and which developed paralysis and died did not die in the same manner as those which had been injected with Jungeblut's acclimated virus obtained from the same source. The incubation period was longer and the course of the disease less abrupt. It is possible that we were not experimenting with the same virulent quotient.

Does poliomyelitis virus change its character as it becomes modified to the mouse; does the virulence of the original virus become enhanced to the point where all animals die even when minute doses are used for the injection; do we deal with a separate virus entirely? These questions cannot be answered by our experiments.

Conclusion. It can only be stated (1) that the specimens sent to us by Jungeblut and Sanders were not neutralized by specific convalescent serums as were other poliomyelitis virus strains and (2) that our present adaptation of Trask's virus in rats does not produce clinical symptoms similar to those which follow injections of Jungeblut and Sanders strain.