by matings of the treated males ($sc^8.Y.B^8/y^2w^ict^6f^1$) to $Y\ sc^{81}\ In49\ sc^8;\ dp\ bw;\ st\ p^P$ females (Table 2). The absence of one wing or leg among the visible mutants was especially striking. Also a few eye mosaics were observed, suggesting that somatic abnormalities occurred concomitantly with the germinal havoc detected. Only a few translocations were recovered.

Comparison of the mean transverse diameter of puffs (*5*) X:2-B; III L:71; III L:72; III L:74; and III L:75 of treated and untreated chromosomes showed possible diminution in size resulting from treatment with this RNA virus (Table 3). However, these differences in preliminary results are not significant. Inoculation, into chicks, of material extracted from progeny of flies exposed to Rous virus so far has failed to elicit avian tumors.

Several reports of induction of tumors in mammalian tissue by this virus (originally discovered to affect fowls) have appeared (*6*). Our studies offer proof of more distant transgeneric effects of Rous virus. The well-known role of insects as vectors of disease and the possibilities offered by use of drosophila in studies on the mode of action of oncogenic and other viruses in tumorigenesis and mutagenesis are thought to be of general interest. The analytical methodology available with drosophila suggests that study of the behavior of viruses, helpers, and hybrids (*7*) in metazoan cells may be approached quantitatively and more precisely now that an oncogenic virus is known to interact with the dipteran genome.

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1. Drs. Bryan, Stevenson, Moloney, and others at the National Cancer Institute have been kind enough to make the virus used in these experiments available.
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Seizure Discharges Evoked in Vitro in Thin Section from Guinea Pig Hippocampus

Abstract. *A thin section prepared from guinea pig hippocampus produced, in chloride-free medium, a train of seizure discharges in response to a single shock applied to the section. Generation of these discharges was ascribed to the lack of inhibitory processes in an absence of chloride ion.*

Eccles and his associates demonstrated that the inhibitory postsynaptic potential (IPSP) in the spinal motoneuron was brought about by an increase in permeability of the membrane to chloride ion, and the same ionic mechanism has been found to play a main role in generating IPSP's in the higher centers as well (*1*). It is deduced, therefore, that if chloride ion is removed from the extracellular space of the brain, the IPSP's are either changed to the depolarizing potentials or abolished (*2*). Thus, because of a lack of inhibitory processes, the brain neuronal network may be brought into such a highly excited state that it tends to generate the seizure discharges. Although this is an interesting surmise, it has not been tested because it is not possible to remove chloride ion completely from the extracellular space in the in vivo experiments. Recently, it has been found that mammalian brain tissue can exhibit electrical activity even if it is excised from the brain and maintained in a chemically defined medium (*3*). Since in this experimental situation chloride ion in the extracellular space can be readily removed, it is possible to test whether the seizure discharges are generated in an absence of chloride ion. Actually, in our experiments, which were carried out on a section from the guinea pig hippocampus, a single shock applied to the slice evoked a train of seizure discharges in the chloride-free medium.

After stunning the guinea pig by a blow on the back of the neck, the brain was removed and divided sagitally along the midline. The surface of the hippocampus facing the thalamus was exposed by removing the brainstem. A slice 0.35 mm thick was prepared from the exposed portion of the hippocampus with a razor blade and a slicing guide, in the same way in which a slice from the cerebral cortex is usually made (*4*). In Fig. 1B, the approximate portion of the hippocampus from which the slice was obtained is schematically shown by the dotted line. The slice was unfolded on nylon mesh with its cut surface upward; it was incubated at 37°C in glucose-saline medium saturated with

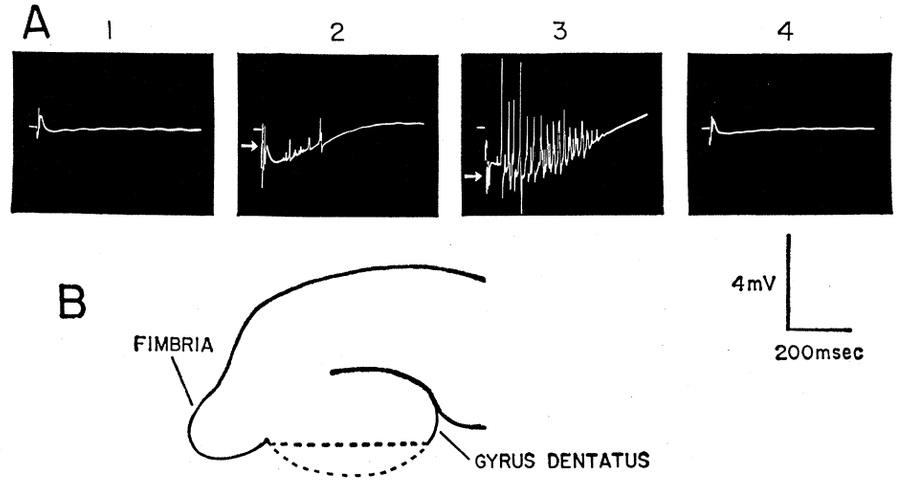

Fig. 1. Generation of seizure discharge in chloride-free (propionate) medium. (A-1) Recorded in normal medium; (A-2) 8 minutes after changing the solution to medium containing chloride at a concentration of 13 m*M*; (A-3) 8 minutes after immersing the slice in chloride-free medium; and (A-4) after returning the slice to normal medium. Note that the seizure discharge in record 2 is much smaller than it is in record 3. Arrows in records 2 and 3 indicate sharp deflections evoked with a short latency. (B) Approximate portion of the hippocampus from which the slice was taken is shown schematically by dotted line.

95 percent oxygen and 5 percent carbon dioxide in an apparatus similar to that developed by Gibson and McIlwain (*5*). During the recording of electrical activities, the surface of the medium was lowered to the level of the nylon mesh. A pair of stimulating electrodes, consisting of ball-tipped silver wires, was placed on the slice, and stimulation was carried out with 5- to 15-volt square pulses, 0.4 to 0.7 msec in duration. A recording electrode, also of ball-tipped silver wire, rested on the surface of the slice; a reference electrode was put in the medium. The normal medium consisted of (final concentrations): 124 m*M* NaCl, 5 m*M* KCl, 1.24 m*M* KH_2PO_4, 1.3 m*M* $MgSO_4$, 2.6 m*M* $CaCl_2$, 26 m*M* $NaHCO_3$, and 10 m*M* glucose. To replace all or part of chloride ion, propionate or acetate ions were used.

Record 1 of Fig. 1A depicts a potential evoked in normal medium. A single shock generated a negative wave of about 20-msec duration with a short negative deflection superposed on it. When chloride ion was completely replaced by propionate ion (record 3), the same stimulus provoked a long train of seizure discharges with a latency of 40 msec. Individual spikes which composed the seizure train were 3 to 10 msec in duration and varied in size between 0.2 and 6.5 mv. One or two inflections were found on the ascending phase of some of these spikes. In the medium that contained a 13-m*M* concentration of chloride, amplitude of the seizure discharge was much smaller and its total duration was much shorter than these were in a complete absence of chloride ion (record 2). When chloride-free medium was replaced with normal medium, the seizure discharge disappeared immediately (record 4). Besides the seizure discharge described above, several biphasic sharp deflections were generated with a short latency (arrows in records 2 and 3). These deflections also developed concomitantly with reduction of the concentration of chloride ion and were observed consistently in the experiment cited in Fig. 1, but they could be elicited only occasionally throughout the present series of experiments. The stimulus artifacts were larger in records 2 and 3 than in records 1 and 4, though stimulus strength was kept constant. This seems to be due to the change in conductance of the medium. In several experiments, we tried to provoke the seizure discharge in normal medium by increasing stimulus strength up to five times that used to evoke the seizure discharge in chloride-free medium. However, even with such strong stimulation, the seizure discharge was not provoked in normal medium.

A similar seizure discharge was observed when chloride ion was replaced by acetate ion. The seizure discharge was augmented by a 1-m*M* concentration of sodium phenobarbitone but suppressed at a concentration of 3 m*M*. The discharge train had a long refractory period and propagated slowly through the slice at a speed of approximately 1 cm/sec.

As discussed in the introductory paragraph, generation of the seizure discharge in chloride-free medium may be ascribed to the lack of inhibitory processes in the neuronal network. There remains the possibility, however, that removal of chloride ion decreases conductance of the medium, thus reducing the short circuit of stimulus current by the medium. This might increase the efficiency of stimulation so that a single shock produces a train of seizure discharges. This possibility may be excluded by the finding that even with a strong stimulus the seizure discharges could not be provoked in normal medium.

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Axonal Delivery of Neuroplasmic Components to Muscle Cells

Abstract. *Substances labeled with phosphate-32 and carbon-14 and applied to hypoglossal nuclei in rabbits traveled down the hypoglossal nerves and after several days began entering only the muscle cells of the tongue. Prevention of axonal delivery on one side caused unilateral labeling of the tongue. Labeled substances delivered by extracellular fluids labeled all cells indiscriminately. The axonal conveyance of neuroplasmic components to peripheral cells may provide a basis for trophic influences of neurons on other cells.*

Maintenance of the axons in peripheral nerves depends on the continual delivery of fresh cytoplasm elaborated in the cell bodies (*1*). The cytoplasm, apparently propelled in a peristaltic manner by the axon (*2*), is continually moved out of the cell body and along the entire length of the axon and all of its branches, supplying them with components that are used in axonal maintenance and activity and that are not (or are insufficiently) supplied by other sources, such as blood or other extracellular fluids and Schwann cells, or by synthesis within the axon. The total volume of neuroplasm may be replaced several times each day (*1*).

Interruption of axoplasmic continuity results in Wallerian degeneration of fibers separated from their cell bodies. After an interval that varies with the length of the distal stump (*3*), degeneration or other trophic changes begin in the muscle or other cells innervated by the interrupted fibers; these changes are clearly distinguished from those induced by interruption of impulses (*3, 4*). We have tested the hypothesis that the trophic dependence of a cell on its innervation is, as in the case of the axon, also based on the continual delivery, by the axon, of substances that originate in the nerve cell. We found that substances (labeled with isotopes) in selected nerve-cell bodies are conveyed down their axons, across the junctions, and into the cells that they innervate (*5*).

We labeled hypoglossal and vagal neurons with P^{32}-inorganic phosphate or C^{14}-amino acids by directly applying solutions of these substances to the posterior tip of the floor of the fourth ventricle in rabbits (1.4 to 2.5 kg) ac-