fer-incubated tissue and its absence in the same cell when the tissue was treated with enzyme. Material specifically removed by ribonuclease as described above is conventionally assumed to be RNA (*11*). This pink-staining basiphilic material in the cytoplasm of normal nerve cells as well as that composing the perinuclear ring of injured cells is RNA.

The appearance of a perinuclear concentration of RNA is the earliest and most conspicuous sign of axon injury in these cells. However, other changes similar to those seen in the vertebrate neuron during injury and regeneration were also observed. Swelling of the nerve cell body is apparent 2 days after injury, but not after the 6th day. The normally pear-shaped cells become more spherical when they swell, and the peripheral cytoplasm stains less intensely, as seen by comparing the matched cells in Fig. 1*B*. At about 4 weeks, when regeneration of the peripheral axon is well under way (*14*), the nucleus of the injured cell has shifted to an eccentric position, usually near the point where the axon emerges from the soma. At 18 days the nucleolus has doubled in diameter, and this persists during the course of axon regeneration for as long as 60 days.

The same elements of response to injury and regeneration occur in neurons of both the cockroach and vertebrates. The early dominant response in the cockroach neuron is a dense aggregation of RNA in the perinuclear cytoplasm. This is probably due to a concentration of ribosomes in this region. The primary response of the injured vertebrate neuron is just the opposite, that is, a breakdown or chromatolysis of the dense RNA aggregates in the Nissl bodies. This seems due to a dispersion of ribosomes from their densely packed sites on the endoplasmic reticulum of the Nissl bodies (*15*). Soon after axon injury, the nerve cell is apparently preparing for the massive increase in synthesis of RNA and protein synthesis associated with later regeneration of its process (*1*). It is curious that increased protein synthesis is preceded by the formation of cytoplasmic RNA aggregates in cockroach neurons, while in the neurons of vertebrates it is preceded by the dissolution of such aggregates. However, in the later stages of axon regeneration in both the cockroach and the vertebrate the nucleus shifts to an eccentric position, and the nucleolus increases in size.

A slight concentration of RNA appears in the perinuclear cytoplasm of uninjured ganglion cells (*12*), as might be expected, owing to normal metabolic activity. However, the exaggerated perinuclear ring of RNA is confined to cells whose axons have been injured. This ring therefore provides a specific marker for linking a given nerve cell body to a single peripheral axon and the muscle that it innervates. We are constructing a cell-body map of the ganglion incorporating this type of information. It should prove useful in examining the neuronal organization underlying specific behavioral acts in insects.

The relatively small number of cells and their high degree of symmetry in opposite halves of the ganglion provide the ideal situation for controlled studies involving RNA–protein synthesis in central neurons. Individually matched cells can be used as experimentals and controls, thereby overcoming the great variability in RNA content seen within normal populations of vertebrate central neurons (*4*). This approach may be promising in examining possible RNA–protein changes associated with learning in an isolated cockroach ganglion (*8*).

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Mosquito Transmission of a Reticulum Cell Sarcoma of Hamsters

Abstract. *A transplantable reticulum cell sarcoma with leukemic manifestations can be transmitted from one hamster to another by means of a mosquito,* Aëdes aegypti *(L.). The transmission seems to be by a transfer of tumor cells, and not by passage of some other oncogenic agent.*

There is good evidence that the contagious reticulum cell sarcoma TM (*1*) of hamsters is transmitted by direct cellular implantation (*2*). When passed by subcutaneous transplantation, tumor cells appear in the blood after 5 days and just before the death of the hamster reach a concentration greater than 100,000 per mm^3. The transmission of TM by the transfer of these circulating cells from tumor-bearing hamsters to hamsters without tumors through the mosquito *Aëdes aegypti* (L.) was indicated in the following experiments.

In one series of experiments, mosquitoes were allowed to feed on tumor-bearing and tumor-free Syrian hamsters. In each of several tumor-free hamsters, one mosquito was implanted subcutaneously by trochar and crushed. Of 26 animals in which the mosquito implanted had fed up to 6 hours previously on a tumor-bearing hamster, 24 developed tumors. Of seven control animals in which the mosquito implanted had fed on a tumor-free hamster, none developed tumors.

In another series of experiments, TM was transmitted to tumor-free animals by subcutaneous implantation of tumor tissue. Fourteen to fifteen days later, the abdomen of each animal was shaved; each animal was then anesthetized with Nembutal intraperitoneally and strapped to a board. When a sample of blood had been taken from the orbital region for a white cell count, each animal was placed individually in a cage of mosquitoes (females, 9 to 10 days after emergence). When a mosquito had partially fed, the feeding was interrupted and it was

Table 1. Transmission of TM from tumor-bearing to tumor-free hamsters by *Aëdes aegypti*. In blood of normal hamsters the white cell count is about 6000.

Donor		Recipients		
No.	White cell count	Total No.	No. developing tumors	Time tumors observed (days)
1	146,850	3	0	
2	111,000	3	0	
3	45,000	12	0	
4	122,000	3	0	
5	65,000	11	2	13,20*
6	82,000	10	2	20,23†
7	159,000	8	1	23‡

* Mosquitoes caged with recipients 35 minutes and 60 minutes, respectively. † Mosquitoes caged with recipients 90 minutes and 50 minutes, respectively. ‡ Mosquitoes with recipient 30 minutes.

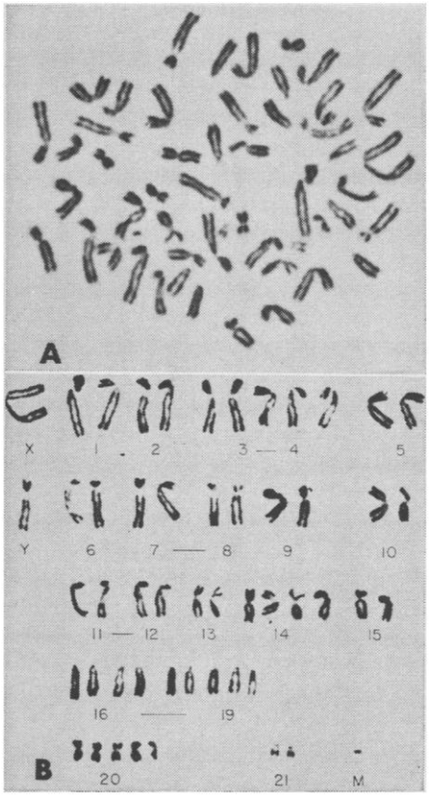

Fig. 1. Metaphase plate (*A*) and karyotype (*B*) of typical cells from tumor arising in a hamster bitten by mosquitoes fed previously on a TM-bearing hamster. The karyotype is identical to that previously described for TM (*2*). The minute marker chromosome (*M*) is shown, X-chromosome, extra chromosome in group 3-4, extra chromosome in group 16-19, two extra chromosomes in group 14-15, and three extra chromosomes in group 20. Total chromosome number is 51. The numbers given in the karyotype correspond to the pairs of chromosomes of the normal Syrian hamster karyotype, which contains 44 chromosomes.

transferred to a cage containing a tumor-free recipient weanling hamster, similarly shaven, anesthetized, and strapped to a board. At least three, and usually five or six mosquitoes, were allowed to feed on the recipient. The time that the last mosquito was introduced into the recipients' cage and the time the mosquitoes were removed were recorded. Seven hamsters were used as donors and 50 as recipients. The results are shown in Table 1.

Within 23 days five of the 50 recipients developed tumors which resembled histologically those of the donors. The first tumor that we observed developed in a hamster that had been bitten by mosquitoes 13 days previously. Two tumors appeared as subcutaneous nodules with metastases, one over the sternum and one over the right abdomen. In three other hamsters, there were no skin or subcutaneous tumors but there was extensive visceral involvement which included mesenteric fat and lymph nodes, kidneys and retroperitoneal nodes, thymus, diaphragm, lungs, and liver. In one hamster, the tip of the sternum was infiltrated.

Chromosome studies were performed on one of the tumors arising from a mosquito bite (*2*, *3*). The karyotype of the cells of this tumor (Fig. 1) was identical to that previously described for TM by Cooper *et al.* (*2*). Of 19 cells examined, 17 contained 51 chromosomes, including a single X chromosome, three extra chromosomes in group 20 (Fig. 1), two extra chromosomes in group 14-15, and one extra chromosome in each of groups 3-4, and 16-19 (*4*). A characteristic minute marker chromosome was also present.

The tumor, TM, used in these experiments has been examined repeatedly for virus by means of tissue culture, passage of cell-free material in animals, and electron microscopy, but no virus has been found. Cooper *et al.* (*2*) have shown that TM has a very consistent and highly specific karyotype, differing from the normal pattern for hamster cells. This karyotype is maintained if the tumor is transplanted to other animals, is induced by feeding tumor tissue, or is passed by caging tumor-bearing and tumor-free animals together (cannibalism). The study of Cooper *et al.* thus indicates that transmission is the result of implantation of tumor cells. In the present study, since the same specific karyotype was maintained in the recipient, the transmission of the tumor by the mosquito is considered to be the result of transfer of viable cells by the mosquito from one animal to the other.

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Quinine-Resistant Plasmodium berghei in Mice

Abstract. *During induction of chloroquine resistance,* Plasmodium berghei *developed resistance to quinine administered in doses near the maximum amounts tolerated by mice. Resistant parasites did not form malarial pigment. Normal sensitivity to both quinine and chloroquine returned and pigment formation resumed during serial passage of the parasites through untreated mice.*

The chemotherapy of human malaria is being complicated by increasing evidence (*1*, *2*) of resistance by parasites to the main groups of synthetic suppressive drugs: 4-aminoquinolines, acridines, biguanides, and pyrimethamine. In contrast, unequivocal resistance to quinine has not been demonstrated in human malaria. Although variable amounts are required to cure *Plasmodium falciparum* malaria, quinine has proved in controlled studies (*2*) to be effective against several strains of *P. falciparum* that show resistance to one or more of the synthetic drugs. Hence quinine is regaining, at least for certain strains, the prominent position it filled before the