In electrochemical pH measurement, as mentioned earlier, Desicote has been of considerable help. Water-repellent glass electrodes and liquid junctions having smooth exterior contours, carrying no liquid from one solution to the next, can materially improve speed and accuracy in rapid pH work. Electrodes of the standard sizes can be used for micro work by suspending a drop between the tip of the glass electrode and the liquid junction; although it adheres to the water-repellent surfaces, the drop will confine itself to a small volume. In working with concave glass electrodes designed for micro work, and also with small electrodes in small containers, Desicote prevents the solution from creeping up the sides of container and electrodes and thus exposing itself to evaporation and absorption of atmospheric gases, with attendant changes of temperature and pH. A similar action is achieved, with associated increase of accuracy, when a glass electrode is used in measuring the pH of a flat surface. The use of Desicote in minimizing electrical leakage on the surfaces of electrode stems and resistors of all sorts (glass, ceramic, silica, polystyrene, and certain other resins) is indispensable in situations where high humidity or even a saturated atmosphere would otherwise deposit a continuous conducting film of moisture upon the surface in question. Desicote is also of value in preventing potassium chloride incrustations from creeping along the outer surfaces of calomel electrodes. In the rapid precision testing of pH buffers, where relative accuracy of .001 pH is desired, study of the behavior of the best liquid junctions has shown that equilibration following change of pH is more rapid and more reproducible when the outer surface of the liquid junction is water-repellent.

One further application, not studied in this laboratory but repeatedly mentioned in the literature (*10–13*), consists in the use of silicone water-repellents in blood needles and syringes and other vessels intended to contain blood, for the prevention or delaying of clotting.

The above is a by no means complete and, obviously, not entirely original summary of the uses of organosilicon water-repellents; the basic uses have already been described by the manufacturers of the primary materials (*8, 9*) and others. However, the wide applicability of water-repellent glassware in every analytical and experimental laboratory has not previously, to my knowledge, been pointed out and is evidently not generally known. I believe that the introduction of these substances to all laboratory workers is worth while in view of the savings of man-hours that can be effected through their use.

References

1. HUNTER, M. J., *et al. Ind. Eng. Chem.*, **39**, 1389 (1947).
2. NORTON, F. J. *Gen. Elec. Rev.*, **47**, (8), 6 (1944).
3. BURKHARD, C. E., *et al. Chem. Revs.*, **41**, 97 (1947).
4. JOHANNSON, O. K., and TOROK, J. J. *Proc. IRE*, **34**, 296 (1946).
5. PATNODE, W. I. U. S. Patent 2,306,222 (1942).
6. NORTON, F. J. U. S. Patent 2,386,259 (1942).
7. POST, H. W. *Silicones and other Organic Silicon Compounds.* New York: Reinhold (1949).
8. *Dow Corning Bull.* J-22.
9. *Gen. Elec. Bull.* CDR-2, CDR-57, and CDR-5.
10. JAQUES, L. B., *et al. Can. Med. Assoc. J., Proc.*, **55**, 26 (1946).
11. PATTON, T. B., WARE, A. G., and SEEGERS, W. H. *Blood*, **3**, 656 (1948).
12. RICE, W. G. *Am. J. Clin. Path.*, **18**, 752 (1948).
13. TURNER, R. D., *et al. Ibid.*, **19**, 296 (1949).

Manuscript received April 25, 1951.

Multiplication of *Rickettsia tsutsugamushi* and Ornithosis Virus in Transplantable Mouse Tumors

Tsuguo Kuwata

Department of Bacteriology,
Chiba University School of Medicine,
Chiba, Japan

Avian pest virus (*1*), vaccinia (*2*), and a few neurotropic viruses (*3–6*) have been reported to interfere with neoplasms. However, several of the other viruses (*7*), including *lymphopathia venereum* (*8*), propagate well in tumor cells with minimal effect upon them. In this paper the influence of *Rickettsia tsutsugamushi* (AGANO-X strain[1]) and the ornithosis virus (KAM strain[2]) (*9*) upon tumors is examined. Tumors tested[3] were the quinone-induced carcinoma (*10, 11*) and fructose-induced sarcoma (*12, 13*). By use of a trocar, both these tumors were implanted in the back of albino mice weighing about 12 g. They developed to a size of 2.0 × 1.0 cm diam 10 days after transplantation and killed the host in 3 weeks.

Experiments with Rickettsia tsutsugamushi. Five to seven days after tumor transplantation, liver suspensions of mice infected with *Rickettsia tsutsugamushi* (10^5 LD_{50}, measured by the intraperitoneal route) were injected directly into carcinomas. Thereafter, development of the tumors was observed, and the mice were sacrificed and the infectivity of tumor and liver

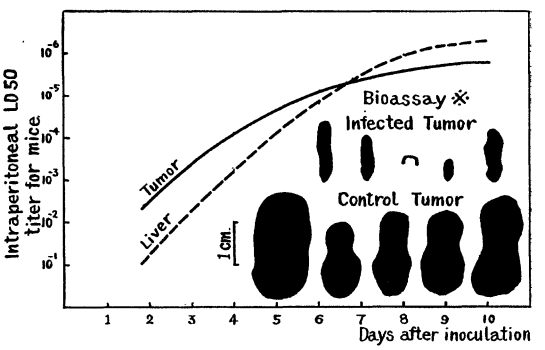

FIG. 1. Infective titer of tumor and liver tissue from mice after inoculation with *Rickettsia tsutsugamushi.*

* Bioassay of rickettsia-infected tumors was made 8 days following inoculation, and results were estimated 11 days thereafter.

[1] Isolation from the patient occurred at the endemic area of tsutsugamushi disease in Niigata, Japan.

[2] Obtained through the courtesy of Geoffrey Rake, Squibb Institute for Medical Research.

[3] Kindly supplied by Nobujiro Takizawa, Department of Pathology, Chiba University School of Medicine.

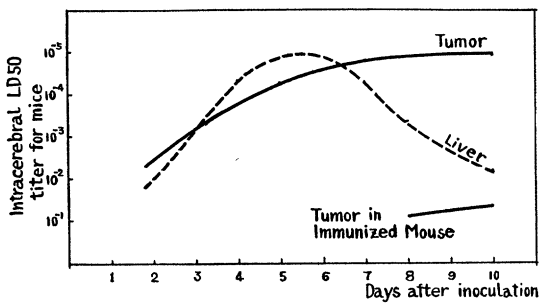

FIG. 2. Experiment with the ornithosis virus.

was measured each 2 to 3 days. One of the typical experiments is shown in Fig. 1. Infective titer of the tumor was higher than that of liver at the first stage of infection, and then both became almost equal at the eighth day after inoculation. Infected tumors developed almost equally with the normal one, probably because it takes some time for propagation of rickettsiae. Bioassay of these heavily infected tumors was performed, and they were found to have minimal ability to develop in 2 weeks. In some instances no growth of the tumor occurred after transplantation. Mice that received transplants infected with *Rickettsia tsutsugamushi* died from infection about 14 days later. Further transplantation of tumors was not successful. Similar results were obtained with fructose-induced sarcoma.

Experiments with ornithosis virus. Brain suspensions from mice infected with ornithosis virus (10^3 LD_{50}, measured by the intracerebral route) were inoculated into carcinomas in a similar way. In this instance, although virus in the tumor reached $10^{-4.5}$ LD_{50} on the fifth day after inoculation and persisted longer than 10 days without decrease, virus in the liver began to decrease after 7 days (Fig. 2). Bioassay of virus-infected tumors revealed the occurrence of partial interference, though it was less marked than in the experiment with rickettsia. However, serial transplantation of virus-infected tumors failed to continue beyond the fourth generation. Dissociation in infective titer of liver and tumor tissues infected with ornithosis virus may be interpreted from the viewpoint that immunity develops in liver, but not in tumor cells (*14*). When mice were immunized intraperitoneally with the active virus before tumor transplantation, the challenged virus did not propagate in tumors, despite few if any, neutralizing antibodies in serum. Details of this aspect of the problem will be published separately.

Comparative studies of the behavior of various viruses toward neoplasms may throw light on the biological nature of viruses on the one hand and on that of tumors on the other.

References

1. LEVADITI, C., and HABER, P. *Rev. d'immunol.*, **3**, 5 (1937).
2. TURNER, J. C., and MULLIKEN, B. *Cancer Research*, **7**, 774 (1947).
3. MOORE, A. E. *Cancer*, **2**, 525 (1949).
4. MOORE, A. E., and O'CONNOR, S. *Ibid.*, **3**, 886 (1950).
5. KOPROWSKI, H., and NORTON, T. W. *Ibid.*, 874.
6. SHARPLESS, G. R., DAVIES, E. S., and COX, H. R. *Proc. Soc. Exptl. Biol. Med.*, **73**, 270 (1950).
7. MOORE, A. E. *Cancer*, **2**, 516 (1949).
8. SHOEN, R. *Ann. inst. Pasteur*, **60**, 499 (1938).
9. HILLEMAN, M. R., HAIG, D. A., and HELMOLD, R. J. *J. Immunol.*, **66**, 115 (1951).
10. TAKIZAWA, N. *Gann*, **34**, 158 (1940).
11. TAKIZAWA, N., and SUGISHITA, M. *Ibid.*, **40**, 153 (1949). In Japanese.
12. TAKIZAWA, N. *Ibid.*, **34**, 1 (1940).
13. SUGISHITA, M. *Ibid.*, **39**, 26 (1948). In Japanese.
14. RIVERS, T. M., and PEARCE, L. *J. Exptl. Med.*, **42**, 523 (1925).

Manuscript received July 12, 1951.

The Control of Pyruvate Oxidation in a Cell-free Rat Heart Preparation by Phosphate Acceptors[1]

M. Rabinovitz,[2] M. P. Stulberg, and P. D. Boyer

Division of Agricultural Biochemistry, University of Minnesota, St. Paul

The "creatine effect" observed by Belitzer (*1*) was a recognition of the role of secondary phosphate acceptors[3] in the control of biological oxidations. This effect was manifested by a marked increase in the respiration of muscle minces when creatine was added to the incubation medium. The increase, however, could not be observed when the tissue was reduced to a fine dispersion. Since that time, numerous studies have been made on enzyme systems capable of coupling phosphorylation with oxidation, but only recently have preliminary reports appeared showing actual control of biological oxidations by phosphate acceptors in heart (*2*) and liver tissue (*3, 4*). In this paper data are presented showing the requirement of hexokinase and glucose for the maximal rate of oxidation of pyruvate by a washed rat heart preparation.

TABLE 1

STIMULATION OF PYRUVATE AND OXALACETATE OXIDATION BY YEAST HEXOKINASE*

Substrate	Solution of yeast hexokinase (μl)	Oxygen uptake (μl/10 min)
Pyruvate $5 \times 10^{-3}M$	0	27
	5	52
	25	77
		μl/40 min
Oxalacetate $1.7 \times 10^{-3}M$	0	66
	25	135

* The procedure used to obtain the enzyme preparation from rat heart, concentrations of various added substances, and conditions of assay have been previously described (*2*).

[1] Supported in part by a grant from Nutrition Foundation, Inc., N. Y. Minnesota Agricultural Experiment Station contribution 2677, Scientific Journal Series.

[2] Present address, Division of Biochemistry, Medical School, University of California, Berkeley.

[3] Creatine and glucose may be considered as secondary phosphate acceptors, whereas adenosine diphosphate (ADP) is a primary acceptor.