

indicate in parentheses that it differs from the diffusion coefficient by a factor of  $10^4$ ; thus they fail to point out the important distinction between them. And they state,

In spite of its larger molecular size carbon dioxide diffuses through tissues 20 to 30 times faster than oxygen does, owing to the higher solubility of  $\text{CO}_2$ .

This sentence leaves the impression that solubility somehow imparts a 20 to 30-fold higher diffusivity to the heavy  $\text{CO}_2$  molecule. Actually the diffusivity of  $\text{CO}_2$  is  $1.378 \text{ cm}^2/\text{day}$  (3), which, compared with 1.607 for  $\text{O}_2$ , is inversely proportional to the square root of molecular weight, in agreement with Graham's Law. Krogh himself implies that the diffusivity of  $\text{CO}_2$  is many times higher than that of  $\text{O}_2$ , for he states (1, p. 273),

... the carbon dioxide produced in the tissues can always be eliminated by diffusion into the capillaries, since the diffusion constant for  $\text{CO}_2$  in tissues is some thirty times higher than for oxygen. The  $\text{CO}_2$  pressure difference between any point in the tissue and the blood must, moreover, in all circumstances, be an absolutely negligible quantity.

Actually, the concentration gradient required to achieve a given  $\text{CO}_2$  transport must be higher than for the same  $\text{O}_2$  transport; therefore Krogh's use of the words *absolutely negligible* indicates that he failed to distinguish between his diffusion constant and the diffusion coefficient. Furthermore, both Prosser *et al.* and Krogh state that the diffusion constant for oxygen increases about 1 percent/deg C, taking the  $20^\circ\text{C}$  rate as unity. This statement is false. It is approximately true for the diffusion coefficient [the temperature coefficient of  $\text{O}_2$  diffusivity in water, as is indicated by the dropping Hg electrode, is 1.6 percent/deg C in the neighborhood of  $20^\circ\text{C}$  (5)], but the influence of solubility, which decreases with rising temperature, will offset the increase in diffusivity. Thus the magnitude of the diffusion constant for  $\text{O}_2$  in water at  $30^\circ\text{C}$  is about  $1.30 \times 10^{-3} \times 0.026 \times 10^4 = 0.338$ , which is almost identical to the value at  $20^\circ\text{C}$ .

These quotations and computations show that Krogh's diffusion constant has been erroneously regarded as an index of diffusivity, and that many biologists have been led to believe that  $\text{CO}_2$  has a higher diffusivity than  $\text{O}_2$  in aquatic mediums. The error has resulted from the unfortunate use of tension units (6) in Krogh's diffusion constant.

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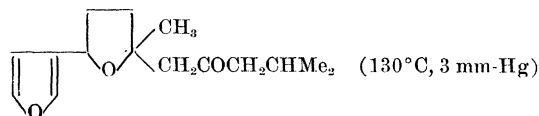
#### References

1. A. Krogh, *The Anatomy and Physiology of Capillaries* (Yale Univ. Press, New Haven, 1936).
2. L. P. Hammett, *Introduction to the Study of Physical Chemistry* (McGraw-Hill, New York, 1952), p. 199.
3. H. A. Spoeher, *Photosynthesis* (Chemical Catalog Co., New York, 1926), p. 84.
4. C. L. Prosser *et al.*, *Comparative Animal Physiology* (Saunders, Philadelphia, 1950), p. 211.
5. W. M. Manning, *Ecology* **21**, 509 (1940).
6. J. Verduin, *Science* **118**, 254 (1953).

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## Antibiotic Effect on *Ceratostomella fimbriata* of Ipomeamarone, an Abnormal Metabolite in Black Rot of Sweetpotato

It has been reported that the respiratory increase in black-rotten sweetpotato roots infected by *Ceratostomella fimbriata* is caused by the uncoupling action of ipomeamarone (Ip.) (1-3)



that had been accumulated in the infected parts by the sweetpotato. It is important, we suppose, from the phytopathological point of view, to learn whether Ip. disrupts the phosphorus metabolism of the penetrating fungus or not, and to make clear the relationship between the accumulated Ip. and the resisting power of the sweetpotato root. M. Hiura has proved that the germination of *C. fimbriata* is controlled in the Ip.-containing culture medium, and we confirmed that the growth, sporulation, spore formation, and respiration of the fungus were restrained by Ip.

In our experiments with the spore-cell suspension taken from the shaking culture, as well as with the mycelium prepared from surface culture, we observed that Ip., even in a low concentration, prevented the absorption of inorganic phosphate by *C. fimbriata* from the medium and the conversion from inorganic P to acid-soluble-organic P and insoluble P. At the same time Ip. promoted spore respiration in the same concentration. The fact became more evident in an experiment using a medium containing  $\text{P}^{32}$  in which Ip. prevented the conversion of inorganic  $\text{P}^{32}$  into acid-soluble P and insoluble P.

S. Spiegelman *et al.* (4) proposed from their data on phosphate metabolism of yeast that ATP, generated through glycolysis or respiration, was required when inorganic P in medium was converted into acid-soluble-organic P and insoluble P; thus uncouplers such as DNP, azide repressed considerably the conversion of inorganic P.

The mechanism of antibiotics such as Aureomycin, usnic acid, gramicidin, and dehydroacetic acid (5), and others has been known to be based on the uncoupling action of oxidative phosphorylation. Now we suppose that Ip. also takes part in the resistive power of sweetpotato root against *C. fimbriata* as an uncoupler.

In addition, oxidative product of chlorogenic acid was observed to repress the oxidative phosphorylation of sweetpotato particles. The oxidative product of chlorogenic acid by polyphenol oxidase in the infected tissues also might be explained to be a factor contributing to the resistance of sweetpotato root.

A further investigation of ours has been focused upon the relationship of Ip. to the nucleic acid metabolism of *C. fimbriata*, the mutation, and adaptation of the fungus.

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#### References and Notes

1. M. Hiura isolated the substance from the infected part and T. Kubota *et al.*, ascertained its structure.
2. M. Hiura, *Sci. Repts. Gifu. Agr. Coll. Japan* **50**, 1 (1943).
3. T. Kubota and T. Matsuura, *Proc. Japan Acad.* **28**, 198 (1952).
4. M. D. Kamen and S. Spiegelman, *Cold Spring Harbor Symposia* **13**, 151 (1948).
5. M. Nomoto and M. Namiki, report to the annual meeting of the Agricultural Chemical Society of Japan, 1 April 1954.

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### Chemopallidectomy: An Investigative Technique in Geriatric Parkinsonians

In March 1954 I described a simple technique of intracerebral procaine injection in the region of the globus pallidus in hyperkinetic disorders. This technique permits one to place a small caliber cannula or catheter into the brain through a trephine opening *without* the use of a stereotaxic instrument. Then, by injections of small amounts of procaine, one can locate that intracerebral area, the procainization of which will temporarily relieve parkinsonian tremor and rigidity, in the contralateral extremities, without causing motor weakness. It was suggested at that time that this technique might be used to locate a physiologic landmark, the permanent destruction of which

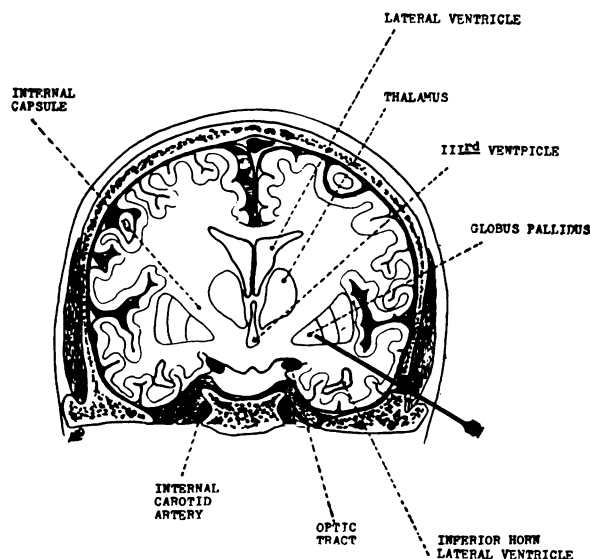


Fig. 1. Cross section of the brain demonstrating the route by which a polyethylene catheter is introduced transcerebrally into the region of the globus pallidus.



Fig. 2. Roentgenogram which was made during chemopallidectomy. Note air in the lateral ventricles and third ventricle. A tantalum stylet which lies within the polyethylene catheter denotes the position of this instrument. Compare with Fig. 1. Injection of procaine through the polyethylene catheter in this case alleviated contralateral tremor and rigidity. Subsequent injection of absolute alcohol produced alleviation of tremor and rigidity persisting for several months and up to the time of this report.

would provide longer lasting relief of tremor and rigidity. The purpose of this communication is to report an investigative effort in that direction.

Using essentially the technique described earlier (1), a small polyethylene catheter with a tantalum stylet is introduced into the brain in the region of the globus pallidus (Fig. 1). Roentgenographic confirmation of the position of the catheter is obtained (Fig. 2). Procaine is injected in increments of 0.25 ml or less, at 5-min intervals, with minor corrections of catheter placement when necessary, until contralateral tremor and rigidity have been relieved. This relief of tremor and rigidity indicates that the "physiologic landmark" has been reached. The catheter is secured so that it remains at this depth and roentgenographic documentation is again obtained. One milliliter of absolute alcohol is then introduced into this area in increments of 0.07 ml every 30 sec. The catheter is left in place for 48 or 72 hr so that the neurolytic lesion can be enlarged if tremor or rigidity recur during this time. Roentgenographic verification of the position of the catheter is obtained before reinjection is carried out.

This technique is currently being investigated in patients older than 55 yr who are considered too old for the operation of anterior choroidal artery occlusion (2, 3). No mortality or lasting motor weakness has yet been encountered with this technique. Of five cases followed for 6 mo or longer, two had recurrence of symptoms in less than 3 mo; three subsequent patients have demonstrated relief of tremor or rigidity or both for 6 mo or more following this procedure.