is attempted in the present study through observations on central bouton changes incident to known lesions in the central and peripheral nervous systems. The data on which the present computations are based have been presented in previous reports.[3,4,5] These data include: (1) determinations of cell size, number and surface area in the various main nuclear groups; (2) quantitative estimates of the number of boutons adjacent to individual cells, to cell groups and in the entire cord; (3) establishment of criteria of bouton alteration under pathological influence which include alterations in number (both increase and decrease) and alteration in morphology; (4) establishment of circumstances dictating the variable changes including direction of influence, effect of age, autolysis and technique, the part played by the duration of the pathological process, the fundamental nature of the pathology, and the bearing cell size may have on the effect; and (5) the establishment of patterns of bouton change peculiar to specific lesions or cord conditions. With this information at hand it is possible to relate alterations in specific locations to known lesions and deduce certain interrelationships which are presented in Table 1.

The restrictions imposed by dependence on chance lesions in human material have necessitated limiting these observations to only the main nuclear groups and their connections. Work is in progress as material comes in which suggests more explicit sources for the groups included in the sensory and internuncial cell columns and also in the somatic efferent columns. It should be pointed out that the quantitative data from control groups at all ages varies plus or minus 5 per cent. necessitating caution in interpreting 0 per cent. values which have been established through comparison with control averages. These values mean only that no change in the synapses at the designated locations is detectable with the technique used.

JEFF MINCKLER

ST. LOUIS UNIVERSITY SCHOOL OF MEDICINE

GROWTH STIMULATION OF PEAS BY TETRACHLORO-PARA-BENZOQUINONE, A FUNGICIDAL SEED PROTECTANT[1]

THE first strictly organic, non-metallic compound to show much promise as a plant protectant against fungous diseases was tetrachloro-para-benzoquinone. In field tests made on pea seed during the past three years, it has very effectively prevented seed decay by soil-inhabiting fungi and has usually induced better yields than metallic compounds of equal fungicidal potency (cuprous oxide, hydroxymercurichlorophenol and ethyl mercury chloride). In tests made under conditions where there was no seed decay, it was the only treatment that increased the yields. The plants from pea seed treated with tetrachloro-para-benzoquinone yielded 9 to 22 per cent. more than untreated controls.

The treatment apparently stimulated the peas in many field plots since the plants were brighter green in color, made more terminal growth and frequently had stronger stems. Such field observations can not be accepted as proof of stimulation because a fungicide such as tetrachloro-para-benzoquinone might promote growth by preventing any one of several diseases of the seeds and roots. As a matter of fact, it has been found that both it and the metallic fungicides frequently increase the yield per plant by preventing post-emergence seed decay, a deleterious but frequently overlooked aspect of the seed disease problem.

In order to prove that tetrachloro-para-benzoquinone was stimulating the plants, it was considered necessary to test its effect on seed in the absence of disease organisms. Disease-free seed were treated with it and other fungicides and then sown alongside untreated seed in steamed soil in the greenhouse. Plants of the varieties Surprise, Wisconsin Early Sweet, Thos. Laxton, Alderman and Perfection have consistently produced 5 to 20 per cent. more dry matter in a 3 to 4-week growing period when grown from seed treated with it than from untreated seed. The other fungicides did not increase the growth of the plants. The seeds and roots of the controls were as healthy as those in the various treatments.

Typical results were obtained in a test with the variety Surprise, using 5 replications of 100 seed each. After 23 days, the average yield of dry plant tissue was: from untreated seed, 9.6 gm; red cuprous oxide, 9.9 gm; hydroxymercurichlorophenol, 9.7 gm; tetrachloro-para-benzoquinone, 10.9 gm; and yellow cuprous oxide, 9.3 gm. A difference of 0.8 gm was significant at the 5 per cent. point in an analysis of variance. The differences in yield were due to differences in growth rates, since there was almost identical emergence (97 to 98 per cent.) from the various treated lots. On the other hand, when seed from the same lots were planted in soil infested with *Pythium ultimum,* all four fungicides increased the emergence by 23 to 25 per cent. and consequently improved the yields. Tetrachloro-para-benzoquinone gave the largest yield increase under these conditions.

These data obtained under controlled conditions provide a suitable explanation for the results ob-

[3] Jeff Minckler, *Anat. Rec.,* 77: 9–25, 1940.
[4] *Ibid., Arch. Neurol. Psych.,* 45: 44–55, 1941.
[5] *Ibid., Am. Jour. Path.* (in press).
[1] Approved by the director of the New York State Agricultural Experiment Station for publication as Journal Paper No. 513, June 1, 1942.

tained under field conditions.[2,3] Tetrachloro-para-benzoquinone serves in the dual capacity of seed protectant and growth stimulant when applied to pea seed. Like any other good seed protectant it can increase the yield under severe disease conditions by preventing seed destruction. In addition, it can stimulate plant growth even in the absence of disease. From a practical view-point this is tremendously important. Heretofore, seed treatment has been recommended as a form of crop insurance against disease because it is needed in one of every three or four fields. As a growth stimulant, the material should pay dividends in practically every field, irrespective of disease conditions. It is of interest to note that the need for such a dual treatment has been previously recognized, and attempts have been made[4] to secure it by using a mixture of a fungicide and various plant stimulants.

It is not known how many species of plants will give the same response to tetrachloro-para-benzoquinone as peas. There is good circumstantial evidence from field observations that lima beans[5] and sweet potatoes[6] may be stimulated, but positive proof has not yet been provided. These field tests and the experiments described herein were made with the commercial product sold under the trade-name of Spergon, which contains one per cent. of a buffering agent. It has been shown that the growth stimulation of peas is dependent on the fungicide and not the buffering compound.

These observations on growth stimulation are of considerable fundamental significance, since they reveal a promising new field of study into fungicides. Further study is needed on the various halogenated members of the quinone series to see if they have properties similar to tetrachloro-para-benzoquinone and whether the growth stimulation and fungicidal properties are dependent upon one and the same chemical grouping within the compound. There is an outside possibility that the growth stimulation comes from decomposition products of tetrachloro-para-benzoquinone, since it is relatively unstable in alkaline media such as some of the soils used in these tests. Regardless of the exact relationship of chemical structure to biological responses, these observations point to a field of study in which both plant pathologists and plant physiologists are vitally concerned.

George L. McNew
New York State Agricultural Experiment Station, Geneva

[2] G. L. McNew, *The Canner*, 92 (6): 56, 1941.
[3] E. G. Sharvelle and B. F. Shema, *Phytopath.*, 31: 20, 1941.
[4] H. E. Croxall and L. Ogilvie, *Jour. Pomol.*, 17: 362, 1940.
[5] G. L. McNew, *The Canner*, 94 (17): 10, 1942.
[6] O. H. Elmer, *Plant Dis. Rep.*, 26: 44, 1942.

SCIENTIFIC APPARATUS AND LABORATORY METHODS

THE BIOLOGICAL APPLICATION OF FOCUSED ULTRASONIC WAVES

High intensity ultrasound above a frequency of 500 kilocycles can best be produced through driving a quartz crystal to expand and contract along its x-axis by an alternating current which is timed to the same frequency of the crystal. The lethal effects of such supersonic waves were first carefully studied by Harvey and Loomis[1] 14 years ago. They used a 50-kilowatt power source and produced rapid death of bacteria and red blood cells in suspensions and of frogs. Since that time many biological and industrial applications of supersonic waves have been found, such as the sterilization of solutions, the homogenizing of oil-aqueous mixtures and of molten metals which would otherwise be immiscible.[2] In all these applications the sound waves have been allowed to proceed in parallel paths from the flat surface of the quartz crystal generator and no effort has been made to increase their intensity and local effect by bringing them to a focus. The authors' studies have utilized the curved quartz crystal method of Greutzmacher,[3] developed in his physical laboratory, and applied it with various improvements and adaptations to the production of focal changes in non-living as well as in living biological material.

The apparatus is essentially an 835 k.c. radio transmitter which provides the radio frequency current for driving the focusing crystal in the ultrasound generator. Ordinary 110 v. 60-cycle current is transformed, rectified and fed into the plate circuit of the first or oscillating stage of the radio frequency generator. This is kept stable, and all tuning is eliminated by introducing into the circuit a master control crystal which has been ground to exactly the same natural frequency as the ultrasound crystal, namely, 835 k.c.[4] After passing through a final amplifying

[1] E. N. Harvey and A. L. Loomis, *Biol. Bull. Marine Biol. Lab.*, 55: 459, 1928.
[2] Ludwig Bergmann, "Ultrasonics and Their Scientific and Technical Applications," p. 264. Translated by H. Stafford Hatfield. New York: Wiley. 1939.
[3] J. Greutzmacher, *Z. Physiol.*, 96: 342. 1935.
[4] This circuit was designed by A. E. Miller, Piezo-electric Laboratories, North Bergen, N. J.