the root cultures was continued for 24 days. There were 4 replicates or a total of 8 plants to each treatment, and each plant, whether treated on the rooting or fruiting zone, was exposed to the same dosage of labeled calcium.

Leaves and sections of stems and fruiting organs of plants were sampled at periods of 3, 8, and 50 hr, and 9 and 24 days. Samples of leaves and stems were from lateral branches, since the peanut plant has a short upright central stem. Gynophores and fruits were thoroughly washed with a stream of distilled water when harvested. Samples were immediately weighed and subsequently dried at 70° C. The plant material was ashed and the calcium precipitated as the oxalate (*7*), which was collected on filter paper over a uniform area for measurement of total calcium by weight and radioactivity with a thin mica window Geiger counter (*5*). Self-absorption and decay corrections were made in the usual manner (*4*).

The pattern of labeled calcium distribution was similar for all plants of a given treatment. Fig. 2 illustrates the uptake of Ca^{45} in μg/gm of dry weight of portions of two representative plants after 9 days. Plants received the complete nutrient solution on the rooting and fruiting zones, and experimental conditions other than the zone of labeled calcium supply were the same in both cases.

Ca^{45} administered to the roots could be detected after 3 hr in all vegetative portions of the plant, being most pronounced in the stem, but was found only in the young peg (gynophore and ovary) of the different fruiting stages. The young peg consistently showed a more active absorption of calcium than the other classes of fruit; however, the capacity for calcium absorption decreased as the peg elongated and entered the fruiting medium. At 9 days the shell of fully developed green fruit contained a very small amount of labeled calcium, but never more than a trace could be detected in the seed. This relationship persisted throughout the 24 days.

The uptake of labeled calcium by the developing fruit was reversed when it was administered to the fruiting zone. The very active absorption of Ca^{45} by the gynophores and by shells and seeds of the fruit from the external supply in the fruiting medium very greatly exceeded the small amount of absorption by these organs when labeled Ca was supplied to the rooting zone of the plant. This emphasized the capacity for calcium absorption by the developing fruit from the external supply in the fruiting medium.

It is noteworthy that when labeled calcium was applied to the roots of the plant, the capacity for calcium absorption by the young peg decreased as it developed as shown by the decreased absorption of labeled calcium. Thus it appears that the calcium content of the young peg was diluted as it increased in size and that most of the Ca^{45} found in the fruit was absorbed in the early stages of peg development. This suggests that the capacity for calcium absorption by the gynophore and ovary is limited after a definite point during their development and that further fruit development is apparently dependent upon the external supply of calcium in the fruiting zone.

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The Influence of Delta and Gamma Benzene Hexachloride upon the Oxygen Uptake of Brain[1]

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During a study of the pharmacology of some stereoisomers of benzene hexachloride (BHC) in mammals, it was observed that whereas the gamma isomer is a potent convulsant agent, the delta isomer is a central nervous system depressant. Moreover, it was found that the two isomers are mutually antagonistic in their effects upon the central nervous system of the rabbit and dog (*5, 6*). In the same studies attempts were made to mitigate the effects of the gamma isomer by the administration of *i*-inositol, which was reported by others to prevent the growth-inhibiting action of the gamma isomer upon certain strains of saccharomyces (*4*). In no instance did *i*-inositol modify the course of convulsant action of γ-BHC. It was believed possible that this failure might be due to lack of penetration of *i*-inositol into susceptible brain cells. In view of these observations, and the often mentioned correlation (*1–3, 7–9*) between narcosis and brain oxygen uptake, the following study was initiated.

In exploratory studies rats received intraperitoneally sufficient drug to produce central stimulation (100 mg/kg of γ-BHC) or central depression (500 mgm/kgm of the delta isomer). The rats were decapitated after the onset of symptoms, and the QO_2 of brain slices was determined manometrically. By this method no difference could be observed between control and test brain slices. For the experiments reported herein, the following technique was employed: Normal rats were killed by decapitation, and the brains were removed and weighed. The entire brain

[1] In previous reports the authors used the word "Gammexane" as a synonym for the gamma isomer of benzene hexachloride. It has been brought to our attention that the name "Gammexane" is a trade mark designation of the Imperial Chemical Industries, Ltd. The term gamma benzene hexachloride has been approved by the Committee on Insecticide Nomenclature of the American Association of Economic Entomologists (*J. econ. Entomol.*, 1947, **40**, 280).

[2] The technical assistance of Anne M. Kunkel and Dorothy E. Stabile is gratefully acknowledged.

was then placed in a homogenizer tube and 0.05 cc of a 10% solution of the drug in peanut oil per gram of brain was added. Pure oil was added to controls. The brain and the oil solutions were then homogenized. Sufficient quantity of a modified Krebs phosphate solution (containing 0.1% glucose) was added to make the final homogenate, containing 125 mg brain (wet weight) in 3 cc. This technique was necessitated by the low water solubility of the BHC isomers. The homogenate was equilibrated with room air at 38° C for 10 min and allowed an additional equilibration period of 10 min after the vessels were closed. Readings were taken at 15-min intervals for a period of 1 hr.

TABLE 1

EFFECT OF ISOMERS OF BENZENE HEXACHLORIDE ON BRAIN OXYGEN UPTAKE

Compound	Brain (mg/gm)	No. of determinations	QO_2 (wet wt)	Ratio experimental/control
Control	....	9	0.79 ± 0.1*	1.00
Alpha	2.5	6	0.92 ± 0.07	1.16
	5.0	6	0.97 ± 0.07	1.23
Gamma	5.0	11	0.76 ± 0.06	0.96
Delta	0.5	4	0.93 ± 0.10	1.18
	1.25	5	1.13 ± 0.09	1.43
	2.5	5	1.23 ± 0.13	1.56
	3.75	5	1.46 ± 0.07	1.85
	5.0	11	1.40 ± 0.16	1.77
Delta + Gamma	5.0 + 5.0	4	1.41 ± 0.19	1.78
Delta + Inositol	5.0 + 25.0	1	1.35	1.71
Gamma + Inositol	5.0 + 25.0	1	0.77	0.97

* $\sqrt{\frac{\Sigma d^2}{N}}$

The QO_2 values obtained on rat brain homogenates containing added drugs are shown in Table 1. Whereas γ-BHC failed to alter the oxygen uptake of the brain, the delta isomer significantly increased this function. Inositol did not alter the oxygen uptake produced by either isomer and γ-BHC exhibited no antagonism against the effects of the delta isomer.

The most striking fact obtained from this study was the unexpected position of the two isomers with respect to their effects upon the brain-homogenate respiration. The narcotic isomer (delta) produced as high as an 80% increase in respiration over the control. Moreover, its pharmacological antagonist, the gamma isomer, a powerful convulsant, did not modify this effect. In addition, *i*-inositol was similarly without effect. The "stimulating effect" of the delta isomer upon the respiration of brain homogenate in the face of its narcotic properties *in vivo* is not yet understood and requires further study. The possibility that this increased oxygen consumption is merely a reflection of the oxidation of the halogenated hydrocarbon cannot be overlooked. However, it is interesting to consider these facts in light of the previously mentioned hypothesis of the mechanism of action of certain narcotic agents.

The unaltered QO_2 values observed in the exploratory studies on rats poisoned prior to decapitation is also noteworthy. Similar results have often been observed for other anesthetics which depress the oxygen uptake of the brain on direct addition *in vitro*. Quastel (7) explains this by an outward diffusion of the drug from the cells to the medium. It does not seem likely that this explanation would hold in the case of the isomers of BHC since these compounds are oil rather than water soluble. No explanation for this divergence can be offered at this time.

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Color Change of Strawberry Anthocyanin with D-Glucose[1]

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Strawberries (garden varieties) are usually prepared by mixing with granulated sucrose prior to freezing. Due to the scarcity of this sugar during the war years, experiments to substitute other sweetening agents were initiated. When crystalline glucose was used a violet-to-blue coloration developed on freezing, which reverted to the original red color on thawing. This color change with glucose may be peculiar to strawberries, since it has not been observed with small fruits such as red raspberries. Interest in the applied as well as in the fundamental aspects of this phenomenon led us to an investigation.

Customarily, sliced strawberries are frozen with sucrose in a ratio of four parts of fruit to one part of sugar. In this preparation no change in color occurs. However, thirty varieties of strawberries grown in Geneva all turned violet when frozen with ordinary commercial crystalline glucose at −23.3° C. in a four to one

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