is raised and makes circling movements, and the forelegs are stretched (Fig. 1).

Sometimes the cat makes beating movements with its forelegs, indicating alternate increase and decrease in muscle tone. Especially, the tone of the back may be very low. About 30 percent of the animals have a marked nystagmus. The cat does not answer to calls for several hours. It is able to walk, but its movements are inaccurate, it falls easily, and uncoordinated movements occur. After 1 to 2 hours the cat calms down, but the clawing movements persist for days. Some cats recover completely after several days. Others fall into a state of complete inertia. They do not clean their fur, and they have sore mouths and sore eyes. They may do several steps in a strange atactic way, and they may take food. Within 8 to 10 days they die. Control injections with Ringer solution have no effect. Injections with Kalbak bouillon sometimes cause, for the first 2 to 3 minutes, light clawing movements; after this the animals are normal. With *Shigella shigae* toxin and with staphylolysin (*10*), the picture under the same experimental conditions is entirely different. Therefore, the picture seems to be typical for streptolysin O. Some of its patterns remind us of a choreic state. Since Sydenham's chorea is related frequently to previous streptococcal infection (*11*), our findings encourage us to undertake further studies.

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Effect of Gibberellic Acid and Indoleacetic Acid on Growth of Excised Fruit Tissue

Gibberellic acid, a metabolic product of certain strains of *Fusarium moniliforme,* is capable of stimulating growth in many plants (*1*). Its effect has been attributed basically to a promotion of cell elongation (*1*), and it has recently been shown to promote cell division (*2*). Auxins, both natural and synthetic, have been found to stimulate callus formation in a wide variety of plant tissues (*3*). Cellular proliferation of excised mature pericarp tissue has been demonstrated in the avocado (*4*). The purpose of this report (*5*) is to show that gibberellic acid is capable of stimulating callus formation in excised fruit tissue cultures and that this effect is greater if indoleacetic acid is supplied simultaneously.

The tissues used in this experiment were obtained from the mesocarp of a mature citron (*Citrus medica*) by slicing the fruit transversely and removing round plugs of tissue with a cork borer. These tissue explants were weighed and planted individually under aseptic conditions on agar medium formulated by Nitsch (*6*), which was modified by replacing yeast extract with a mixture of thiamine, pyridoxine, nicotinic acid, and glycine. The tissue explants, each approximately 2 mm thick and 8 mm in diameter, had an average weight of 115 mg. Various amounts of gibberellic acid (*7*) and indoleacetic acid were added to the base medium so that the final concentrations of 0, 0.5, 5.0, 25, 50, and 100 ppm of gibberellic acid and 0, 0.1, and 1.0 ppm of indoleacetic acid were obtained. This gave a total of 18 combinations. The final medium was dispensed at 10 ml per 6-dram screw-top vial and autoclaved at 15 pounds pressure for 20 minutes. At least eight cultures were used in each combination except for the two highest gibberellic acid concentrations, each of which consisted of five cultures.

From Fig. 1 it is apparent that both indoleacetic acid and gibberellic acid stimulate callus formation. Microscopic observations at the end of eight weeks indicated that most of the original cells had died. The callus tissue, however, was fully alive and consisted of many relatively small, thin-walled cells. These anatomical observations suggest that any increase in weight must be due to an increase in cell number.

The curves in Fig. 2 represent the change in fresh weight of each tissue explant as the gibberellic acid concentration increased. In the absence of indoleacetic acid, 5 ppm of gibberellic acid caused maximum weight increase, while the higher and lower concentrations used were much less effective. In the presence of indoleacetic acid, 0.5 ppm of gibberellic acid caused a marked increase in weight, and the stimulatory effect of gibberellic acid increased up to a concentration of 25 ppm. Only at 50 ppm and 100 ppm of gibberellic acid was there a decrease in the stimulatory response.

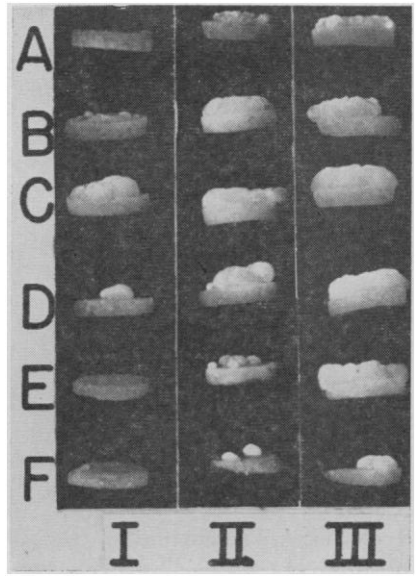

Fig. 1. Side view of citron fruit explants grown *in vitro*: (vertical rows) concentrations of indoleacetic acid (I, 0; II, 0.1; and III, 1.0 ppm); (horizontal rows) concentration of gibberellic acid (A, 0; B, 0.5; C, 5.0; D, 25; E, 50; F, 100 ppm).

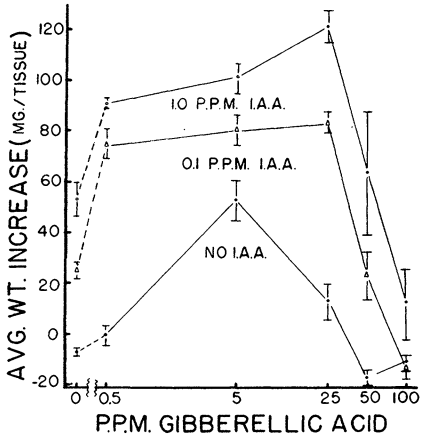

Fig. 2. Average increase in fresh weight of excised citron tissue in relation to gibberellic acid and indoleacetic acid. Gibberellic acid concentrations plotted on logarithmic scale.

This experiment suggests that gibberellic acid and indoleacetic acid stimulate callus formation in excised citron tissue and that the tissues respond to a wider range of gibberellic acid when they are grown in the presence of exogenous indoleacetic acid.

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Vitamin A-Carotene Deficiency Affects Serum Protein and Utilization of Carotene by Steers

A close relationship between serum proteins and vitamin A-carotene utilization or tissue storage, or both, has been shown (*1, 2*). Limited data (*3*) indicate that the cow's carotene requirement is markedly increased after a prolonged suboptimum carotene intake. Therefore, this study was undertaken to determine whether vitamin A-carotene deficiency would influence the serum protein fractions or alter the utilization of dietary carotene by the beef animal.

Eleven 800-lb steers were pretreated as follows: six animals were fed a grain-straw diet (carotene-free), and five steers were fed a grain-dehydrated alfalfa ration, until the former group exhibited vitamin-A deficiency symptoms (about 120 days). All animals were then fed a carotene-free diet for the 14-day experimental period. Pure carotene (10 percent alpha and 90 percent beta in an aqueous solution with Tween 80, *4*) was administered by means of a stomach tube to all steers (15 mg of carotene/100 lb of body weight) on alternate days for the first 10 days of the experimental period.

Table 1 shows the plasma vitamin A and carotene and the serum protein fractions (*5*) of the normal and deficient animals before and after carotene administration. As expected, the plasma vitamin A and carotene increased after administration of carotene to the deficient steers. The normal steers exhibited a decreased plasma level of both carotene and vitamin A, the latter changing only slightly. Prior to carotene treatment, the percentage of beta globulin in the serum of the deficient steers was significantly higher than that in the normal steers. In the pretreated, vitamin A-deficient animals, serum albumin increased (average, 5.6 percent) while alpha and beta globulin decreased significantly (3 and 5.8 percent, respectively) after the 10 days of carotene administration. Although the remaining fractions differ, none were statistically significant. The total serum protein was not affected by either vitamin A deficiency or carotene administration. No correlation was found between the change in blood vitamin A and carotene and the change that occurred among the serum proteins. Although work with human serum (*1*) has shown that beta globulin binds about 50 percent of the blood carotene, the beta globulin was markedly greater in the carotene-deficient steers in this study.

All animals were subjected to liver biopsy (*6*) at 0 and 14 days of the experimental period. The samples were analyzed for vitamin A and carotene (*7*). Table 1 shows the liver storage of vitamin A and carotene, as affected by pretreatment, before and after oral administration of carotene. The results of the liver analyses were extremely consistent within animals with the same pretreatment. Under the conditions of this experiment, there was no increase in the liver carotene content of the deficient or normal steers as a result of carotene administration. However, there was a large increase in the liver storage of vitamin A in the normal steers following carotene administration. There was no increase in the liver vitamin A content of the carotene-deficient animals. This marked difference in vitamin A deposition in the liver may suggest a lack of conversion of carotene to vitamin A in the deficient steers. However, the fact that plasma vitamin A increased in the deficient animals does not appear to uphold this hypothesis. On the other hand, these results may indicate extrahepatic utilization or storage of the vitamin A formed in the deficient animals, or both. This explanation is supported by results with rats (*8*), in which a higher dosage of vitamin A was required for liver deposition in deficient rats than was required to alter the blood level of the vitamin (*9*).

Table 1. Summary of results.

Day	Plasma		Serum*				Liver	
	Vitamin A (μg/100 ml)	Carotene (μg/100 ml)	Albumin (%)	α-Globulin (%)	β-Globulin (%)	γ-Globulin (%)	Vitamin A (μg/g)	Carotene (μg/g)
			Normal steers (average of five steers)					
0	65	240	41.2	17.6	16.6	24.6	23.41	7.36
2	60	263						
4	42	222						
6	48	232						
8	52	202						
10	53	150	44.5	15.4	15.7	24.4		
14							38.55	6.73
			Deficient steers (average of six steers)					
0	2	0	39.4	17.6	21.7	21.3	1.57	2.21
2	10	11						
4	26	22						
6	26	43						
8	28	51						
10	29	28	45.0	14.6	15.9	24.5		
14							0.58	0.54

* Each figure represents an average obtained from figures for four steers.

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