Table 1. Effect of Gibberellin on Growth of Plant Tissue Cultures on Synthetic Medium

Plant source	Part	Type of tissue	Growth value			
			4 weeks		8 weeks	
			Control	Gibberellin (10 p.p.m.)	Control	Gibberellin (10 p.p.m.)
Helianthus annuus	Petiole	Crown gall	12·8	4·4	49·1	11·0
Vinca rosea	Stem	Crown gall	6·3	2·0	35·4	4·3
Nicotiana tabacum	Stem	Crown gall	3·4	3·8	13·5	15·8
Melilotus officinalis	Stem	Crown gall	2·6	2·9	8·9	14·3
Melilotus officinalis	Root	Callus	2·2	3·1	7·0	15·8

These results are only indicative of the results using 10 p.p.m. of gibberellin. Even at this one level, however, it is apparent that no generalizations can be made concerning the effects on plant tissues *in vitro* since both stimulation and inhibition result according to the test material.

A broader account is now being prepared which takes into consideration such problems as the effects of different levels of the gibberellins, the effects of varying the medium and the importance of the type of tissue used as well as its source.

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Effect of Gibberellic Acid on the Growth of Pea Seedlings when imbibed through the Seed Coat

Stowe and Yamaki[1], in a recent review paper, have listed two hundred and two references relating to the isolation and effects of the gibberellins upon various physiological processes in plants.

Work with these plant-growth substances has, however, been primarily concerned with their effects upon plant growth and development subsequent to germination. It is the purpose of this brief communication to demonstrate that gibberellic acid may produce a typical response when imbibed through the seed coat of the dwarf Telephone pea prior to germination.

Approximately fifty dwarf Telephone pea seeds were planted in each of two small wooden flats filled with sterile vermiculite. The flats were placed over porcelainized germination pans so that added solutions could be retrieved after dripping through the vermiculite. Directly after planting, the vermiculite in one flat was saturated with a solution of 1·0 mgm./l. gibberellic acid. The second flat was supplied with distilled water. The solutions caught in the germination pans were poured back over the respective flats daily until utilized or lost. Plants were grown in the greenhouse under long-day (16 hr.) conditions.

Eleven days after planting, treated and control seeds had germinated and were well above the level of the vermiculite. Treated dwarf pea seedlings were, at this time, approximately six times as tall as the control seedlings. The percentage of germination in both flats was comparable.

Seventeen days after treatment the plants in both flats were harvested. The heights of treated seedlings were significantly greater than the heights of control seedlings. The axillary buds, as well as the main axes of the stems, exhibited markedly increased elongation in treated plants. The stems of the treated seedlings were markedly thin as compared with the controls.

Subsequent experiments have revealed that the response to gibberellic acid described above may also be obtained by allowing dwarf pea seeds to imbibe gibberellic acid solution prior to planting.

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Genetic Effects of Carbon Monoxide, Cyanide and Azide on *Drosophila*

The study of chemical mutagenesis has directed attention to the possibility that the so-called spontaneous gene mutations may in fact result from the accumulation of mutagenic substances arising as a consequence of normal metabolism. In bacteria, the spontaneous mutation-rate of dividing cells may be 40 times greater than that of non-dividing cells[1]. On the other hand, there does not seem to be any simple relation between mutation and cell division itself[2]. This may be interpreted to mean that, as a result of metabolic activity associated with synthesis and growth, chemical mutagens arise and may eventually react with the genetic material. The discovery of 'anti-mutagens'[3] would appear to support this hypothesis. An anti-mutagen might directly mop up traces of the mutagen, or it might initiate further biochemical reactions serving to divert the mutagen into alternative channels.

D'Amato and Hoffman-Ostenhof[4] have recently reviewed the possibility that spontaneous mutation in plants may result from the accumulation of mutagenic by-products of metabolism. The genetic effects of respiratory inhibitors are clearly relevant to the problem. Although some inhibitors might prove to be mutagens in their own right, capable of reacting more or less directly with the genetic material, others might operate simply by disturbing normal metabolic activity in such a manner that an excessive production of natural mutagens results, enabling a greater number of 'spontaneous' mutations to be recorded. Cytogenetic analysis of such mutations would give a picture essentially the same as that obtained from the analysis of spontaneous mutations obtained from untreated material. We should also keep in mind the possibility that any genetic effects of respiratory inhibitors might stem from interference with recovery processes. Wolf and Luippold[5] have found an association between oxida-