inal function in retinal ischemia (*10*). However, a profound and prolonged vasospastic response to oxygen could produce the paradoxical situation of localized anoxia in an oxygen-rich environment.

On the other hand, injury due to the inhibition by oxygen of intracellular enzyme reactions could readily account both for the death of visual cells and for segmental lesions in their processes. Oxygen at elevated pressures is a potent toxin for living cells (*11*). Support for the concept that oxygen at high concentrations has a direct toxic action on the intact animal is found in the potentiating action of carbon dioxide upon the neurotoxic effects. Convulsive responses, paralysis, and death are all accelerated or increased in incidence by the addition of this carbon dioxide (*12*).

Preliminary studies in our laboratories (*1*) indicate that the eye lesion due to hyperoxia may be prevented by the use of 98 percent oxygen and 2 percent carbon dioxide at 3 atm. However, with this respiratory gas mixture, there was a shift in the locus of toxic action of oxygen from the eye to the central nervous system which was manifested clinically by overt neurologic deficits and histologically as selective neuronal necrosis. Two separate mechanisms may be operative; the increased vascular resistance induced by hyperoxia may, on the one hand, result in ischemic retinal injury, and on the other hand, protect the central nervous system from the histotoxic effects of hyperoxia. There is much speculation regarding the pathogenesis of oxygen toxicity. If these preliminary findings prove correct, the therapy of oxygen poisoning may be more problematic than hitherto thought.

The perils to the eye posed by oxygen at high pressure are of particular significance to hyperbaric medicine and to undersea ventures. Since, however, prolonged exposure to oxygen at a partial pressure of over 350 mm-Hg is regularly attended by toxic manifestations (*13*) our man-in-space program carries this hazard, too (*14*).

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Auxin and Kinetin Interaction in Apical Dominance

Abstract. *The effects of auxin on the inhibition of lateral buds in decapitated bean plants are enhanced if kinetin is applied together with auxin. The uptake of ^{14}C-indoleacetic acid by the stumps of decapitated plants is increased in the presence of kinetin and leads to extensive transport of ^{14}C-indoleacetic acid in the stems. The increased bud inhibition resulting when auxin and kinetin are applied together may be due to greater amounts of auxin reaching the buds, but an alternative explanation is that metabolites are directed from the buds to the point of hormone application.*

Until recently it was thought that the correlative inhibition of lateral buds is regulated by a single hormone, auxin, produced in the shoot apical region and moving downwards in the stem, where it directly inhibits the growth of the buds. It is now becoming apparent that in bud inhibition, as in other growth phenomena, there is an interaction between the three types of hormones, auxin, kinin, and gibberellin.

Sachs and Thimann (*1*) showed that if auxin is applied to the stump of decapitated pea seedlings, the resulting inhibition of the lateral buds is released if kinetin is applied directly to the lateral buds. Jacobs and Case (*2*) presented evidence that auxin and gibberellin may interact in apical dominance. They found that if gibberellic acid (GA) and indoleacetic acid (IAA) were applied together to decapitated pea seedlings, the buds remained inhibited for a longer period than if IAA alone was applied. They demonstrated that the transport of ^{14}C-labeled IAA in decapitated pea stems is enhanced when GA is also applied, and they suggested that the observed increased apical dominance can be ascribed to the greater amounts of IAA reaching the buds when IAA and GA are applied together. We have observed similar interaction between kinetin (6-furfurylaminopurine) and IAA in apical dominance, but a different interpretation of the results from that of Jacobs and Case is suggested.

Experiments were conducted with seedlings of French bean (*Phaseolus vulgaris*, cv. 'Canadian Wonder') which were grown in "John Innes" potting compost in boxes in a heated greenhouse. The seedlings were grown for 2 weeks and were then decapitated 5 cm above the node of the paired primary leaves. Preparations of IAA and kinetin in lanolin, each at a final concentration of 0.1 percent, were applied alone and in combination to the decapitated internodes. Equal quantities of lanolin, contained in gelatin capsules (about 1 ml), were applied to each plant; in this way the lanolin was applied to the cut surface and to the uppermost 2 mm of the sides of the stump. Measurements were made daily of the lengths of the basal internodes of the buds in the axils of the primary leaves (Fig. 1). Treatment with both IAA and IAA plus kinetin resulted in inhibition of bud growth up to the 5th day. Thereafter the buds of the IAA-treated plants began to grow out slowly, whereas those treated with IAA and kinetin remained inhibited, so that by the 14th day the two treatments had resulted in greatly different growth. The buds of the lanolin controls and of the kinetin-treated plants began to grow out after the 1st day, and they continued

to grow actively up to the end of the experiment. These results are evidence of a synergism between IAA and kinetin in apical dominance in bean plants, when the kinetin is applied with the IAA to the decapitated stump.

One explanation for this effect is that kinetin is in some way increasing the transport of IAA in the plant, as suggested by Jacobs and Case for the interaction between IAA and GA in apical dominance. The following experiment tested this possibility.

Pot-grown seedlings of bean were transferred to a growth room at 20°C under continuous illumination for at least 24 hours; they were then decapitated as before, leaving the primary leaves and the internode above on each plant. Immediately after decapitation, 1 μc of IAA-2-^{14}C was applied to the cut surface of the stump of each plant. After the solution had evaporated or had been absorbed by the tissues (that is, after 3 to 4 hours), the plants were divided into two groups of 12 plants each. Hormone preparations in lanolin paste were now applied in capsules, as described. One group was treated with IAA alone (1 percent) and the other with a mixture of IAA and kinetin each at a final concentration of 0.1 percent. The plants were left for 24 hours under continuous light. After this period stem pieces (6 cm each) were harvested from 10 plants in each group for assay of radioactivity, and the remaining two plants were used for separate treatment as follows.

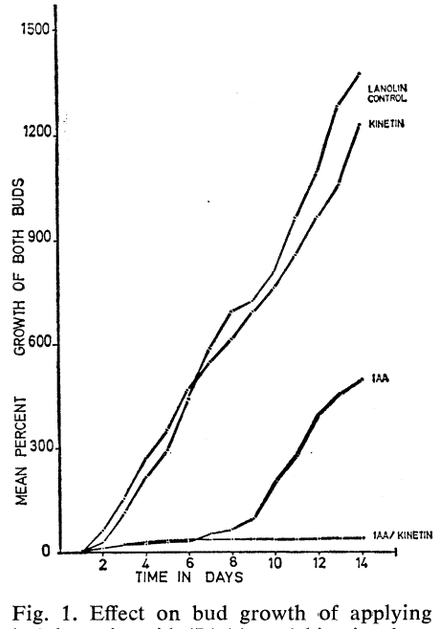

Fig. 1. Effect on bud growth of applying indoleacetic acid (IAA) and kinetin alone and in combination, to decapitated internodes of bean plants. Hormones were applied as preparations in lanolin at final concentrations of 0.1 percent for each hormone, whether alone or in combination. Bud growth is shown as the mean for two laterals in axils of primary leaves (five plants per treatment). The mean lengths (mm) of the buds from base to tip in the respective treatments on the 14th day were as follows: (i) Lanolin, 54.2 $\pm$ 7.9; (ii) IAA, 18.0 $\pm$ 4.5; (iii) Kinetin, 37.8 $\pm$ 7.3; (iv) Kinetin + IAA, 4.6 $\pm$ 0.8. The differences between all treatments are significant.

The stems of plants harvested for assay were each divided into 1-cm pieces; the uppermost piece was discarded, and each of the five remaining pieces were extracted separately with 80 percent methanol. The extracts were reduced in volume and placed on lens paper mounted on planchettes for counting in a proportional counter (Fig. 2). There was greater activity in the stems in plants treated with a mixture of IAA and kinetin than in plants treated with IAA alone. That the two curves are effectively parallel to each other suggests that kinetin affects primarily the uptake of IAA at the point of application rather than the transport of IAA. The remaining two stem pieces from each group were extracted with 80 percent methanol. The reduced extract was partitioned by paper chromatography with a mixture of isopropanol, ammonia, and water (10 : 1 : 1) as a solvent system. The radioactivity in the extracts was confined almost entirely to the peak corresponding to IAA. Thus, the radioactive material was apparently moving in the stem almost wholly as IAA. Further evidence that kinetin may affect the uptake of IAA was provided by the results of an experiment carried out with 2-cm pieces of bean internode, but with essentially the same technique as that described for decapitated plants. There were significantly greater amounts of ^{14}C-IAA in the second 1-cm sections when kinetin was also applied to the apical end than with auxin alone.

Further evidence is necessary to establish whether the effect of kinetin is primarily to increase the uptake of IAA or to stimulate its transport in the stem, but, in any case, greater amounts of IAA were present in the stems when kinetin was added with IAA. Possibly this increase in IAA concentration in the stem was directly responsible for the greater bud inhibition in the plants receiving IAA and kinetin together.

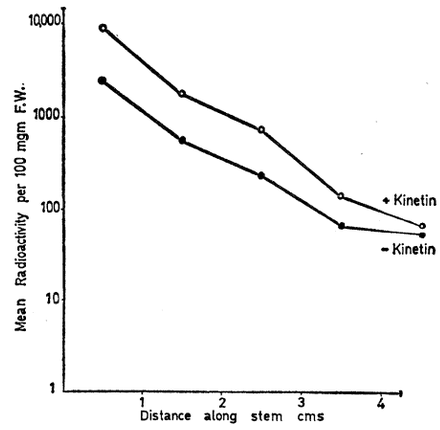

Fig. 2. Effect of kinetin on movement of IAA-^{14}C in decapitated internodes of bean. The IAA-^{14}C was applied as 0.1 percent lanolin preparation to both series, and kinetin (0.1 percent) was also applied to one series. Abscissae indicate distance from point of decapitation (logarithmic scale). The mean counts are based upon ten determinations in each case. Differences between treatments are significant at 5 percent probability level at all distances from site of application.

However, another interpretation is possible. There is a movement of ^{32}P and ^{14}C-sucrose towards the decapitated stumps of pea plants to which IAA has been applied (*3*), and this auxin-directed transport is further enhanced if either kinetin or GA is used in combination with IAA (*4*). Indeed, we have also found that hormone-directed transport is most pronounced when all three types of hormone (IAA, GA, and kinetin) are applied together (*5*). Thus, the increased apical dominance observed when IAA is used in combination with kinetin, or with GA, may be due to the mobilization of metabolites into the decapitated stump and away from the axillary buds, rather than a direct inhibition of the buds by optimum auxin concentrations.

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