The Role of Auxin in Inductive Phenomena

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Abstract. The current status of our knowledge of auxin effects on floral induction is summarized. The most general effect is inhibition, although the concentration of synthetic auxins added to plants tends to be too high for us to be certain that the inhibitory effects are truly physiological. Studies of endogenous levels of auxin have focused almost entirely on IAA-like bioassay activity. Chemical identifications of endogenous IAA are needed and feasible. In addition, a search for other auxins involved in vegetative to floral transitions, their chemical identification, and measurement of their changing levels in the plant are urgently needed.

I have been asked by the organizers of this Symposium to review briefly the current status of research on auxin in the induction of flowering and to suggest possible future avenues for exploration.

It is highly appropriate that this meeting should be held in Czechoslovakia because the first report that auxin could function as a floral inhibitor was that by Dostál and Hošek in 1937. In addition, since 1937 the effect of added indol-3-ylacetic acid (IAA) and other auxins on flowering has been quite thoroughly investigated, with the timing and location of such effects being a special interest of the Institute of Experimental Botany of the Czechoslovak Academy of Sciences.

In the late 1930's and 1940's the failure of scientists to find the hypothesized single flower stimulating hormone drove them to look for other explanations. The idea that auxins were flower-inhibiting substances and that the induction of flowering might involve anti-auxin effects became popular. The easiest path to follow was to add auxin to plants; many such publications appeared. I remember the first such report at national meetings in the USA: a large dose of IAA was reported to inhibit flowering. Solon Gordon asked in his usual polite way if there was any evidence that this large dose stopped all development, thus making the relation with flowering completely nonspecific. The speaker rather shamefacedly admitted that was the case. In addition to such cases of complete developmental inhibition, several cases were found where mM amounts of IAA caused ethylene production, with the ethylene in turn inducing flowering (e.g., the bromeliads). These auxin effects are presumably pharmacological not physiological.

The early research on the effects of added auxin was reviewed by Lang in 1961. Research since then has been summarized in the volumes by Bernier.
et al. (1981). Inhibition of flowering has been the most frequently observed effect of auxin. In most cases the photoinduced leaf seems to be the site of auxin action rather than the later transforming shoot apex. This conclusion is based on both the greater effectiveness of IAA that has been added to the leaf compared to the shoot-tip (e.g., Ogawa 1962) and on timing studies in which IAA inhibits most when given to a leaf just before or during the inductive photoperiod (e.g., Salisbury 1955, Ogawa 1962, Evans 1964) — i.e., before the floral stimulating effect has presumably reached the apex. For SDP Chenopodium, however, IAA added at 0.1 mM inhibited flowering when added to the shoot tip, so long as it was applied close to the time of the inductive photoperiods (Seidlova and Khatoo 1976) and concomitant anatomical studies of the shoot apex convinced them that the added IAA was inhibiting flowering by increasing apical dominance — release from which marked the start of floral differentiation. A nice series of experiments by Krekule and Přívratský (1976) using 14C-IAA confirmed the view that even IAA added to the cotyledon of Chenopodium was actually inhibiting flowering by moving to the shoot tip.

The high concentrations of IAA added in these studies of floral inhibition — typically 0.1 to 1.0 mM — are worrisome even though the volume added is sometimes small. As Salisbury pointed out in his book (1963), such quantities cause wrinkling of laminas and twisting of petioles in treated leaves of Xanthium — effects apparently pharmacological.

To allay this anxiety about the concentrations of IAA or other auxins added, convincing evidence about the extent to which the added auxins exactly substitute for endogenous auxin would be valuable. At present, such evidence seems to be nonexistent. The data that most nearly approach this desideratum came from several papers on the Princeton clone of Coleus blumei, a day-neutral plant. Excising axillary buds and branches caused compensatory growth of the main shoot and also speeded flowering; substituting 1% IAA in lanolin for the axillaries slowed flowering once again, but did not affect the compensatory growth (Jacobs et al. 1959; cf. also Aloni 1976). Other research on this clone provided evidence from auxin bioassays, tracheary regeneration and abscission studies that 1% IAA in lanolin provides auxin into Coleus shoots at a level that matches that provided by the endogenous sources (summarized in Jacobs 1979). However, the inhibition of flowering by IAA was relatively slight (only 7 days later than the 69 days to flowering observed in the control group) (Jacobs et al. 1959).

Studies of endogenous auxin in plants undergoing floral induction have been almost all based on bioassay measurements. Several investigators provided data on plants that were already fully induced and hence were not relevant to our interest in the early stages. The first people to investigate endogenous auxin levels reported auxin activity in their extracts as though it were due to IAA. As separation methods improved, paper- and thin-layer-chromatography was used, with \( ^{2}I\text{IAA} \) being followed (e.g., Bronchard 1957, Gilson 1957). There was, apparently, an unspoken assumption that IAA, and only IAA, was the endogenous auxin.

For other types of plant hormone, for a long time there has been evidence for multiple forms whose relative proportions change as floral development