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Auxin and Red Light in the Control of Hypocotyl Hook Opening in Beans

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ABSTRACT

Evidence is presented to support the suggestion that endogenous auxinlike substances participate in controlling the unbending of the hypocotyl hook of *Phaseolus vulgaris* L. (cv. Black Valentine). An acidic indole was detected in hook diffusates by fluorometry; triiodobenzoic acid, an inhibitor of auxin transport, prevented red light-induced unbending, and indoleacetic acid can be substituted for tissue just above the elbow region as an inhibitor of opening. Indoleacetic acid also stimulated growth of shank cells, and red light increased the sensitivity of this tissue to the hormone. A small red light-induced stimulation of auxin transport through the inside half of the hypocotyl shank was observed and may be related to light-induced unbending of the hook.

During unbending of the hypocotyl hook of bean, growth occurs first in cells at the basal, concave portion of the elbow and in the upper portion of the straight tissue just below (13). This growth is accelerated by red light (5, 13), and elongation of elbow cells apparently depends on the physiological state of the shank cells below (13). In order to understand the mechanism of light-induced stimulation of opening, it is necessary to determine what growth regulators are involved in the elongation of the cells.

Klein *et al.* (9) and Klein (8) have shown that removal of the apex and cotyledons hastened hook unbending, leading these authors to postulate that an auxin-like inhibitor was produced distal to the hook. Other growth regulators have also been implicated in hook opening. Based on the observation that GA counteracted the inhibition of unbending which occurred after the shank was removed, Klein (8) suggested that a substance similar to GA moved up from the shank to stimulate opening. Kang *et al.* (7) and Kang and Ray (6) have pointed to ethylene as a natural inhibitor of bean hook opening since the gas retarded opening at very low concentrations, was produced by the hypocotyl, and its evolution was markedly reduced by red light. A lowering of ethylene production from pea plumules by red light has also been shown by Goeschl *et al.* (3).

The data to be presented here will concern the relationships of auxin to the unbending of the hypocotyl hook. Efforts will be made to determine if auxin can account for the various events occurring during unbending, and if light has any effect on auxin physiology in the hypocotyl.

MATERIALS AND METHODS

Hypocotyl hooks including 5 mm of distal tissue and 3 cm of proximal tissue were excised from 7- or 8-day-old etiolated bean plants (*Phaseolus vulgaris* cv. Black Valentine). Growth was measured with an ocular micrometer after shadowgraphing the tissue; hook angle was determined with a goniometer as described previously (5, 13). The light source (500 erg/cm^2·sec) was a fluorescent tube filtered by red Plexiglas (Rohm and Haas, No. 2444) and red cellophane so that wavelengths below 600 nm were not transmitted (13).

IAA was applied to isolated hooks by incorporating the growth substance into 1.5% agar; the agar was cut into 5 × 5 × 1 mm blocks, and the blocks were applied to the distal ends of the hypocotyls. Triiodobenzoic acid was applied either as a lanolin paste (10%, w/w), or by placing the isolated hypocotyls on their sides in 90-mm Petri dishes containing 10 ml of TIBA[1] solution at the desired concentration. Controls were treated in the same manner but were placed in water. Ten hooks were usually used per treatment, and experiments reported here were performed at least three times with similar results.

In preparation for measuring IAA transport, two groups of five agar blocks were aligned on a microscope slide and separated by a 1-μl disposable pipet. The pipet served to keep the blocks separated during the experiment. A 2-mm hypocotyl section was then placed on two agar blocks so that the basal surface of the inside (concave) half rested on one block and the outside (convex) half of the other. Following placement of five sections on the agar blocks, indoleacetic acid-1-^{14}C (28 mc/mM) at 4 μM was applied to the distal surface in a block of 1.5% agar. After the transport period (2 hr in the dark), the tissue was first weighed, then the hypocotyl tissue, both sets of receptor blocks, and sometimes the donor blocks were counted in Bray's solution by a Packard liquid scintillation spectrometer.

For the collection of diffusible substances, hypocotyl segments consisting of only the basal portion of the elbow and about 1 to 2 mm of shank tissue were placed on filter paper soaked with 5 mM KCN for 15 min. The KCN treatment was used to reduce auxin destruction at the cut surface (15). The basal ends of 20 hypocotyl segments were then placed on 0.5-ml discs of 1.5% chloroform-washed agar. After diffusion in the dark for 2.5 hr, the agar discs were extracted three times with methylene chloride containing 0.1 N HCl. The organic fractions were pooled and partitioned against three 1-ml aliquots of 10 mM phosphate buffer, pH 7.5. The aqueous phases

[1] Abbreviation: TIBA: triiodobenzoic acid.

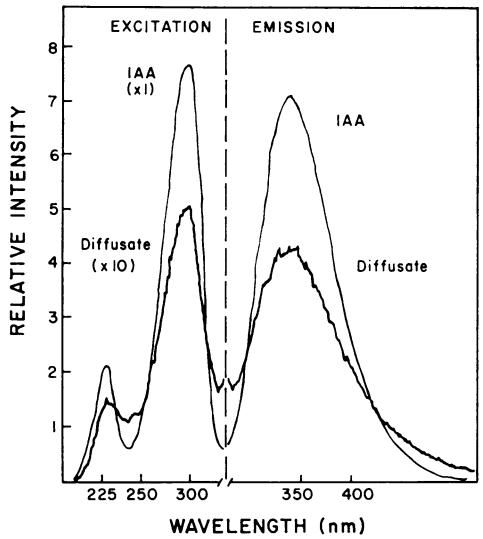

FIG. 1. Excitation and emission spectra of a purified diffusate from the basal portion of 20 hypocotyl hooks. Indoleacetic acid (2 μM) in 10 mM phosphate buffer, pH 7.5, is used for comparison.

Table I. *Effect of TIBA on Light-induced Hook Opening*

Hooks were placed in continuous red light and incubated for 24 hr.

Treatment	Opening
	degrees[1]
Control	85 ± 12
200 μM TIBA (soak)	19 ± 7
20 μM TIBA (soak)	27 ± 6
2 μM TIBA (soak)	61 ± 12
200 μM TIBA (soak) for 24 hr; removed for 28 more hr	121 ± 8
TIBA (10 mg/g lanolin) in ring at base of elbow	34 ± 15

[1] Ten hooks were used for each treatment and the data are expressed as the mean with its standard error.

were then pooled and analyzed in an Aminco-Bowman fluorometer as described by Hertel *et al.* (3). Similar extraction of synthetic IAA in agar resulted in a 60% yield.

RESULTS

Direct evidence for the presence of an auxin-like growth substance utilizing the *Avena* curvature assay has been shown by Kang and Ray (5). Further proof for an IAA-like substance is presented in Figure 1. The excitation and emission spectra of a diffusible substance from the base of the hook elbow are identical to those reported for indole compounds (16). The amounts found for each tissue section are about five times those measured by Kang and Ray (5). Only about 50% or less of the substance could be diffused from the apical surface of the segments during the same 2.5 hr time period.

The involvement of endogenous auxin in the process of hook opening was further investigated by utilizing an inhibitor of auxin transport, TIBA (10). As shown in Table I, concentrations of TIBA as low as 2 μM effectively inhibit light-induced hook opening. This retardation is reversible, however, since removal of the hooks results in a normal amount of opening after 28 hr even in the case of the highest concentration of TIBA. The inhibitor is also effective when applied in a ring of lanolin to the base of the elbow (line 6).

As further evidence for the involvement of auxins in hook opening, IAA could be used to substitute for the tissue located above the basal portion of the elbow. The inhibitory effect of the tip of isolated hypocotyls could be shown most clearly after the tissue below the elbow (the shank) was removed. The presence of the shank alleviated the tip effect (13). IAA was then substituted for tips in the following experiment. All tissue above the top of the hook was cut from isolated hypocotyls and the shanks were also removed from half of them. IAA was then applied to cover the lower half of the upper cut surface; plain agar blocks were used for controls. The data in Table II show that the shanks are able to alleviate most of the inhibitory effect of IAA on hook opening or, in other words, the auxin seems to mimic the effect of the tip by inhibiting most markedly the opening of those hooks which have no shanks.

While IAA seems able to substitute for the tip as an inhibitor of hook opening, the nature of the light-induced stimulation of shank growth has not yet been fully examined. IAA has been shown by Kang and Ray (5) to stimulate shank growth in the light, but only small segments were used, growth at the outer and inner surfaces was not differentiated, and no dark controls were used. The data of Figures 2 and 3 were obtained by applying agar blocks containing various concentrations of IAA to apical ends of 3-cm hypocotyl shanks cut off just below the base of the elbow. Fresh agar blocks were reapplied after 6 hr, and the elongation of three successive 5-mm sections down the inside and outside of the shanks (see inset to Fig. 2B) was measured after 12 hr in darkness or red light.

It can be seen that all portions of the hypocotyl shank, from the upper 5 mm (Fig. 2A and 3A) to the lower 5 mm (Fig. 2C and 3C), grow only slightly without IAA, but growth in the light is still less than that in the dark. When IAA is added, growth of the upper 5 mm at both the inside and outside portion of the hypocotyl (section A) is stimulated, but now, for each concentration of IAA, these upper sections grow somewhat more in the light than in the dark. It is also interesting to note that IAA above 0.3 μM is inhibitory to growth of upper sections on the inside half of the hypocotyl shank in the dark, while inside, upper sections in the light are not inhibited until IAA concentrations exceed 3.0 μM. Though other inside sections (Fig. 2, B and C) also elongate in response to IAA, none show any marked inhibition with higher concentrations, and growth in the dark is almost always greater at each IAA concentration. Growth at the outside of the hypocotyl is not inhibited by IAA at any of the concentrations used.

Comparing the growth data for applied IAA with that of identical sections of intact hooks (arrows at ordinates), it appears that in intact hooks, also, growth of only the upper 5 mm

Table II. *Applications of IAA to Hypocotyl Hooks with Long or Short Shanks*

Hypocotyls were cut at the top of the hook, and IAA or plain agar was placed on the lower half of the distal end. All treatments were incubated in continuous red light for 18 hr.

Treatment	Opening	
	3.0-cm shank	1.0-cm shank
	degrees[1]	
Control	38 ± 5	42 ± 4
IAA (6 μM)	30 ± 8	11 ± 8

[1] Data represent the mean and standard error for each set of 10 hooks.

is stimulated by light, while the lower sections grow more rapidly in the dark. One can mimic, therefore, the effect produced by light on the inside halves of shanks of intact hypocotyl hooks by adding one concentration of IAA (about 0.03 μM) to the shanks alone in both light and dark treatments. Since IAA sensitivity of the upper 5 mm of the hypocotyl seems to be increased by light, the presence of higher levels of endogenous auxin may not be needed to produce the light-induced growth stimulation seen in hypocotyls with elbows intact.

If endogenous auxin regulates the opening of the hook in an inhibitory way, then light must act to relieve this inhibition. The effect of light on auxin movement was investigated by measuring IAA transport through freshly excised 2-mm sections taken from the following areas of the hook: tip sections from 2 mm below the cotyldons, hook sections from the top of the hook, shank sections from just below the elbow. The sections were then cut in half lengthwise so that transport of IAA through the inner and outer halves could be measured separately. It should be mentioned here that for polar transport about 15% of the counts were lost after 2 hr and there was no difference in loss between the various sections. If the receptor blocks were placed at the apical end of the sections and acropetal transport measured, no counts from the donors could be detected in the receptors after 2 hr.

Looking first at the counts (presumably auxin) reaching the receptor blocks (Table III), it appears that more radioactivity is transported through the outer halves compared to the inner in both tip and hook sections. The situation is reversed, however, in the shank sections where over three times more radioactivity is delivered to the inside receptor. When the counts in the receptor are expressed as a percentage of the total taken up by the tissue, it can be seen that for the outside halves more radioactivity is transported through hook sections; transport through the inner half is quite slow for the tip and hook sections compared to that seen in shank sections.

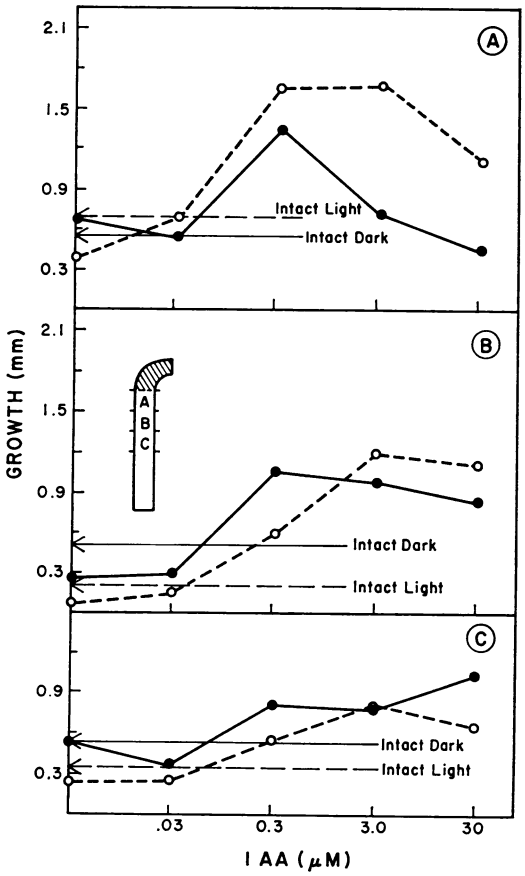

FIG. 2. Growth of three successive 5-mm sections down the inside surface of isolated shanks (unshaded area of inset at B). Dotted lines designate treatments in continuous red light; solid lines are for completely etiolated treatments. Arrows at ordinate refer to growth of similar sections of hypocotyls in which the elbow portion (shaded area of inset at B) was left intact. Each point represents the mean of five hypocotyls.

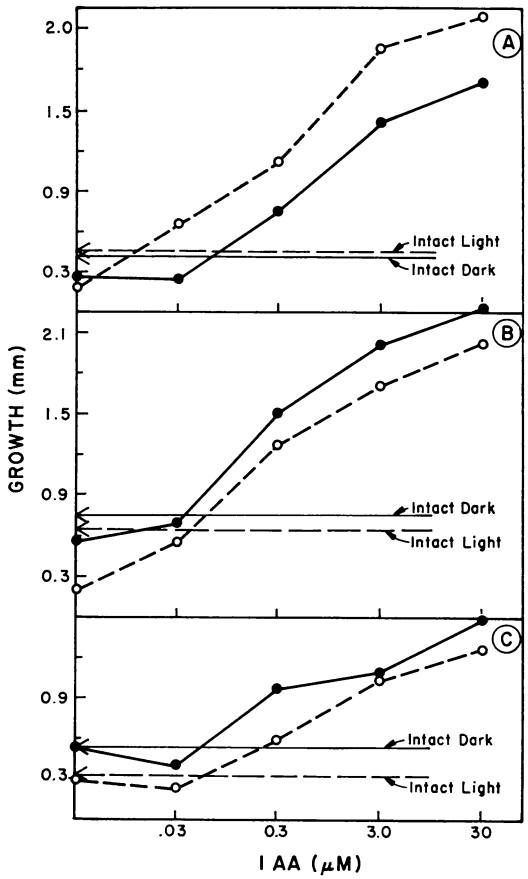

FIG. 3. Growth of three successive 5-mm sections down the outside surface of isolated shanks (unshaded area of inset at Fig. 2B). All symbols are the same as in Figure 2. Each point represents the mean of five hypocotyls.

A possible relationship between red light and auxin transport can be seen in Table IV. Sections 2 mm in length were cut from just below the elbow either immediately after isolation of the hypocotyls from the intact plant (zero-time) or after 10 min red plus 170 min dark or 180 min dark; auxin transport was measured as described in "Materials and Methods." The area used corresponds to the shank sections of Table III, but these were not cut in half longitudinally before measuring transport. Sections from this location were chosen because the area seems to participate in light-controlled opening of the hooks (13) and because it is the area which shows an increased sensitivity to auxin in red light (Figs. 2A and 3A).

From the data for a representative experiment, it appears that an increase in transport over the zero-time controls occurs in the outer half of the sections as a result of the 3-hr incubation period of hypocotyls in darkness or with 10 min red light (Table IV). The transport rate of IAA through the inside half is lower in sections taken from hooks left in the dark for 180 min compared to zero-time controls, but if the hypocotyls from

Table III. *Transport of IAA through the Inside and Outside Halves of Sections Cut from Three Locations on the Hypocotyl*

Sections 2 mm long were taken from 2 mm below the cotyledons (tip), the top of the hook (hook), or from just below the elbow (shank). They were cut in half lengthwise, each half was placed separately on plain agar blocks, and the apical surface was covered with an agar block of 4 μM IAA-1-^{14}C. Transport period was 2 hr in the dark. Each datum represents the average of 10 sections.

Location of Section	Receptor		Tissue		
	cpm	*cpm/mg fresh wt*	*cpm*	*cpm/mg fresh wt*	*Receptor/uptake × 100*
Tip–outer half	25	5.2	212	44.9	10.5
Tip–inner half	20	4.7	189	45.4	9.4
Hook–outer half	48	7.7	292	46.8	14.1
Hook–inner half	34	5.0	313	46.0	9.8
Shank–outer half	52	4.6	711	63.5	6.8
Shank–inner half	165	13.2	652	52.9	20.0

Table IV. *Effect of Red Light on Auxin Uptake and Movement through Shank Sections*

Sections 2 mm long were cut from just below the hypocotyl hook of intact plants or from isolated hypocotyls incubated either in the dark for 180 min or in the light for 10 min followed by 170 min of dark. The sections were placed on two blocks so that the inside half of each section rested on one block and the outside half on the other. A donor block of 4 μM IAA-1-^{14}C covered the upper surface. Transport period was 2 hr in darkness. Each datum represents the mean of 10 sections for the zero time control and 15 sections for the dark and red light treatments.

Time of Cutting	Receptor Location	Receptor		Tissue		Total Uptake	
		cpm	*cpm/mg fresh wt*	*cpm*	*cpm/mg fresh wt*	*cpm*	*Receptor/uptake × 100*
Zero-time	Outer half	121	13.8	1394	79.2	1702	7.2
	Inner half	187	21.2				11.0
180 min dark	Outer half	136	15.4	1177	66.4	1419	9.6
	Inner half	106	11.9				7.3
10 min red + 170 min dark	Outer half	133	15.4	1129	65.3	1391	9.6
	Inner half	129	14.8				9.3

which the sections were taken were exposed to red light for 10 min and then returned to the dark for 170 min, the decrease in transport rate is much less striking. The amount of radioactivity taken up by the 2-mm sections (counts in receptors plus counts in tissue) also decreases if the hypocotyls are isolated for 180 min, but light seems to have no effect. If the transport rate is expressed as a percentage of the total counts taken up, it can be seen that exposure to light prevents more completely the decrease in transport rate seen in inside halves of sections from dark tissue. Or, put another way, the transport rate through the inside half of sections from isolated hypocotyls exposed to light is somewhat greater than through the inside half of those taken from completely etiolated hypocotyls.

The results of six different experiments similar to the one presented in Table IV have been analyzed statistically, and the means expressed as a percentage of the zero-time control (Table V). There is no significant difference between means of the counts in receptors under the outer halves of the sections regardless of whether the hypocotyls had been pretreated with light. Similarly, the counts in the tissue are unaffected by light. Data showing a light-induced stimulation of transport through the inner portion of the sections, however, are significant at the 1% level.

DISCUSSION

The purpose of the experiments reported here was to test the hypothesis proposed by Klein *et al.* (9) and Klein (8) that endogenous supplies of an auxin-like substance control, in an inhibitory way, light-induced opening of the hypocotyl hook. Direct evidence for the presence of an auxin-like substance diffusing from the base of hook sections was presented by Kang and Ray (5). An acidic indole with fluorescence properties similar to IAA (16) is shown here to be present in hypocotyl diffusates (Fig. 1).

The involvement of endogenous auxin is also suggested by substituting IAA for the area above the hook. For example, removal of the apex and cotyledons hastens hook opening, but the original rate is restored with dilute concentrations of IAA (8, 9, 12). IAA substitutes for tissue just above the base of the elbow, as shown in Table II. Further data suggesting a role for auxin in hook opening are found in cutting experiments which point to a polarly transported inhibitory substance (13), and the inhibitions by TIBA (Table I) as well as the stimulations of opening by the auxin antagonists naphthylmethyl sulfide acetate (8) and *p*-chlorophenoxyisobutyric acid (5).

Cytokinins (5, 12) and abscisic acid (5) which are also thought to occur in plants will inhibit opening similarly to IAA, but, unlike IAA, high concentrations are needed and even then the effect is only partial; the strictly polar movement of cytokinins is also open to dispute (2, 11). At this time, though, it is impossible to rule out these substances as endogenous regulators of hook unbending.

Ethylene has been implicated as a naturally occurring inhibitor of bean hook opening (6, 7); it is evolved by isolated hypocotyls, and the rate of evolution decreases in tissue exposed to red light. If red light only acted to reduce ethylene evolution and had no effect on endogenous auxin, one would not expect the inhibitions of opening in the light by TIBA (Table I) or inhibitions when the shank is removed or when a cut is made only on the inside half of the shank below the elbow (8, 13).

An explanation for the light-induced reduction of ethylene evolution may be the effect of light on internal supplies of auxin at the elbow region. Endogenous ethylene evolution has been shown to be controlled at least in part by endogenous auxin (1); since the source of auxin (distal tissue) is removed when the hypocotyl is isolated, and if the auxin level is de-

Table V. *Cumulative Means of Six Experiments Designed to Test the Effect of Red Light on Auxin Movements*

Each experiment, set up as described in Table IV, consisted of three replications for sections cut from either dark- or red light-treated hypocotyls and two replications for sections cut from intact plants (zero time). Five sections were used for each replication. The values for the treatments were then expressed as a percentage of the zero time control, and the significance between the means of six experiments was determined by the Student's *t* test. Means in each column followed by unlike letters differ significantly at the 1% level.

Time of Cutting	Receptor Locations		Tissue
	Outer half	Inner half	
180 min dark	116a	65b	89d
10 min red + 170 min dark	112a	79c	90d

creasing more rapidly in the light than in the dark, lower rates of ethylene production would result. Thus, ethylene may in fact be an endogenous regulator of hook opening, but its evolution is controlled by the effects of light on endogenous auxin.

Concerning growth substances which stimulate hook opening, it is unlikely that GA-like substances move up from the shank to the elbow cells under the influence of red light (5, 13). GA does stimulate opening somewhat, however, and can overcome the inhibition of 0.2 M 2-chloroethyl-trimethylammonium chloride, but GA is ineffective in the dark, leading Kang and Ray (5) to conclude that light does not change internal levels of GA.

It is actually not necessary to postulate the involvement of another growth stimulatory substance during hook opening since, as first shown by Kang and Ray (5) and expanded here (Figs. 2 and 3), an increase in sensitivity to IAA can account for the growth increase seen in the shank. Furthermore, if the elbow cells are considered to be inhibited by endogenous auxin, a reduction of the auxin level could account for elongation seen in the base of the elbow.

The necessity for the shank for alleviation of the tip (13) or IAA-induced (Table II) inhibition may be due to an accumulation of auxin at the cut proximal surface; when this surface is close to the elbow as is the case when the shank is removed, the elevated auxin levels would inhibit cell elongation (Fig. 2A) and thus hook opening. Evidence that auxin does indeed accumulate at the cut surface of bean hypocotyls even when they are on agar has been reported by Zaerr and Mitchell (17). An alternative explanation would be that cutting results in an inactivation of endogenous auxin, leading to both retarded growth and reduced hook opening. Thus, while the involvement of other growth regulators with hook unbending should not be excluded, the properties of auxin (ability to stimulate growth and regulate ethylene production) can account for many of the events seen in the hypocotyl during this time.

Since endogenous auxin would retard hook opening, light may act by alleviating the auxin-induced inhibition of growth. It is unlikely that light exposure leads to auxin destruction, since a stimulation of shank growth is always observed after red light (5, 13), and the data in Figures 2 and 3 show that auxin is necessary for growth of the shank tissue. Furthermore, no effect of light on loss of ^{14}C from IAA was detected during the transport tests (unpublished data). A light-induced synthesis of auxin is equally unlikely because the growth of elbow cells is already inhibited by auxin within the tissue. This supposition is corroborated by the partial induction of hook opening by auxin antagonists (5, 8). Light does not seem to act by changing the sensitivity of the elbow cells to auxin, since applied auxin can halt unbending even after the hooks have begun to open (6).

Thus, endogenous auxin levels must decrease in order for elongation to occur and a likely mechanism would be a more rapid removal of the auxin after exposure of the hypocotyl to light. This suggestion is supported by the inhibition of hook opening after treatment with TIBA (Table I), an inhibitor of auxin transport (10). Furthermore, growth of the hypocotyl is accelerated by light and growth seems to be correlated with auxin transport (Table III; Ref. 4). It should also be noted that since the hypocotyl hook is constantly opening in the dark (accompanied by hook reformation due to asymmetric growth of the tissue above) (13), tissue at the top of the hook will be located just below the elbow in about 12 to 24 hr. Therefore, the faster rate of auxin transport through the inside halves of shank sections compared to hook sections (Table III) can be called an increase in auxin transport accompanying opening.

Evidence that red light may in fact accelerate transport rates (or at least prevent a steady decline) is shown in Tables IV and V. Kang and Ray (5) were unable to find light-induced differences in diffusable auxin, but in some experiments they bioassayed for growth substances 6 to 8 hr after cutting the hook; by that time a significant portion of the auxin diffusing through the tissue could have accumulated at the cut surface (17), not reaching the agar receiver at all. Furthermore, Kang and Ray (5) used the whole hook for their studies and were thus measuring the lowest of many different transport rates (see Table III). Failures to observe light effects on auxin diffusion after only 2 to 3 hr (5) may also be due to the use of the entire hook region, since without the shank, unbending is quite slow (8, 13), and changes associated with hook opening may be very small and occur after the diffusion period. The use of radioactive IAA in the present study, the 2-hr transport period, the employment of 2-mm sections from well defined areas of the hook, and the collection from either inside or outside halves greatly increased the sensitivity of the method.

If one considers only the transport through inside halves of the sections in Tables IV and V, there is a correlation between light-induced growth and auxin movement. There is also a relationship between the decreased growth observed after the hypocotyl is isolated (13) and a reduced rate of uptake and transport through the inner half of the section. The differences between the inside halves of light and dark treatments, though statistically significant, are quite small, so a relationship between light and auxin transport remains to be proven. It can be pointed out, however, that only relatively small differences in growth rate between two sides of an organ can result in a change of angle (14).

A generalized sequence of events from light exposure to hook opening is now tentatively proposed. Light may act upon the upper shank in such a way that sensitivity to endogenous auxin and hence growth is stimulated. This may involve an acceleration of auxin transport. Events in the upper shank could then lead to an alleviation of the auxin-induced inhibition (presumably due to auxin-regulated ethylene production) of elbow cells resulting in their growth and ultimate opening of the hook. An apparent dependence of the growth of elbow cells on the shank cells immediately below has already been pointed out (13).

In order to further test the relationship between light and endogenous growth regulators, one must look more closely at the effects of red light on auxin transport. It is also very important to understand the forms of auxin in the elbow cells and which forms directly control elongation. Growth of the upper shank is dependent upon a supply of hormone and continues for at least 12 hr even when the tip which supplies the auxin is removed (Fig. 2A, arrows at ordinate); since the rate of auxin transport through the hypocotyl is 5 to 7 mm/hr (unpublished data), it follows that all the auxin in the elbow cells should move beyond the growing region of the shank within 3 to 4 hr. Because it is unlikely that more auxin is synthesized in the hook, the endogenous hormone must be in such a state that it can be fed gradually into a transport stream. The solutions to these problems should clarify our understanding of how opening of the hook is enhanced by red light.

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