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The Transportable Auxin Pool[1]

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R. K. DE LA FUENTE[2] AND A. C. LEOPOLD
Horticulture Department, Purdue University, Lafayette, Indiana 47907

ABSTRACT

Evidences from experiments with stem sections of sunflower seedlings suggest that the transport of auxin may be limited by a restricted pool size of transportable auxin and restrictions in the availability of transport sites. A steady state of transport is observed over a range of lengths of stem sections, and over a wide range of auxin contents. The capacity of the sections to transport a pulse of auxin declines with aging after cutting, 50% decline occurring at about 10^+ hours; the transportability of a pulse of auxin declines rapidly after the completion of uptake, 50% decline occurring at about 1 hour. A chase treatment with unlabeled auxin does not alter transport, but a pretreatment with auxin depressed subsequent transport for about 1 hour. In depleted tissues such pretreatment is not inhibitory but rather is promotive of transport. The interpretation offered is that transport is limited by the pool size and transport sites, and roles for these factors are suggested in relation to the auxin transport gradient and the tropistic responses.

Historically, the physiological picture of the auxin transport system has progressed from a qualitative description of transport (its polarity, velocity, and distribution through the plant) toward increasingly quantitative studies (the kinetics of transport, the phases of entry and exit from the tissue, interactions with inhibitors or other auxins). The quantitative analysis of transport was enormously facilitated by the advent of isotopically tagged auxin (*e.g.*, 7); it has become evident that auxin transport is an active transport in the classic sense (6, 14, 16), that it is apparently a secretive process (2, 11) presumably taking place at the cell periphery—probably at the plasmalemma (3, 15, 18). These developments present us with the possibility of asking quantitative questions about the site of auxin transport and the pool of auxin filling these transport sites. By a temporal separation of the loading and unloading of the tissues with auxin, we have attempted to describe the transportable auxin pool and to make inferences concerning the site of transport.

METHODS

Plants of sunflower (*Helianthus annuus*, L. var. Russian Mammoth, purchased from Vaughan Seed Company) were grown in flats of vermiculite in a growth chamber (2000 ft-c, 16-hr photoperiods, 22–25°) to an age of 8 to 10 days (longer periods where indicated). Sections were cut from the hypocotyl, ordinarily 6 mm in length beginning 1 mm below the cotyledonary node. The sections were placed upright on plain agar (1.0%) in a plastic box through which humidified air is continually passed. The auxin, indoleacetic acid-1-^{14}C (specific radioactivity 10.4 mc/mmole, purchased from New England Nuclear) was introduced by placing the sections apical end down on a 2.5-cm circle of agar containing 10^{-5} M IAA-1-^{14}C for 3 hr unless otherwise noted. In cases where there was a delay between the loading of the section with auxin and the unloading, the sections were placed apical end down on plain agar for this interval. Unloading of the transportable auxin was obtained by placing the basal ends of the sections on a plain agar disc (2.5-cm diameter, 2 mm thick) to allow diffusion of the auxin into the agar for 2 hr except where otherwise noted.

The amount of radioactivity in the agar receptor discs was determined by placing the disc on filter paper of the same size, inserting into a glass vial with 5 ml of 95% ethanol, and shaking for 10 min to remove the auxin from the agar. Without removing the filter paper and partially dehydrated agar, the vial was dried in a water bath at about 50°. After drying, 1 ml of methanol was added to take up the auxin, this was shaken, and then 10 ml of scintillation counting solution (PPO and POPOP in toluene) were added. The samples were counted for 5 min in a Packard TriCarb scintillation spectrometer. A counting efficiency of better than 80% was obtained. The radioactivity in the tissues was divided into a soluble and an insoluble fraction by grinding the 20 sections in 8 ml of 95% ethanol, filtering through Whatman 3 paper, and placing the liquid filtrate in one vial (soluble fraction) and the residue on the paper in another vial (insoluble fraction). Fractions were dried, taken up in 1 ml of methanol, and then assayed by scintillation counting. The data for soluble and insoluble counts in the tissue are added together and reported here as radioactivity in the tissues. Each treatment was carried out with 20 hypocotyl sections in each of three replications.

Recutting to remove the basal cut surface of the section was carried out for all experiments except where noted. Ordinarily this involved reducing a 6-mm section to 4 mm by removal of the basal 2 mm of tissue just before the radioactive donor was applied.

RESULTS

It is known that auxin transport from excised sections declines after 2 hr or more of transport (7, 20, 24). Suspecting that this decline might be due to deteriorative changes at the cut surface of the tissue, we attempted to alleviate this temporal decline by recutting the sections at the basal or apical ends, or both. Sunflower hypocotyl sections 4 or 6 mm long were cut and aged for 24 hr, after which time one or two slices were removed from either or both ends, reducing the section to 4 mm. Donor blocks were applied for 2 hr, followed by receptor blocks for 2 hr, and the data in Table I show that, whereas uncut sections lose 89% of their ability to deliver auxin to the receptor block (unaged sections transported 3056, aged sections 340 cpm), recutting the basal end of

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[2] Present address: Biological Sciences Department, Kent State University, Kent, Ohio.

the section reduced this loss to 45%; recutting the apical ends had little effect on transport but consistently depressed the amount of auxin in the tissues. As a standard procedure in experiments involving aging, then, we recut the base of each section before the donor block was applied.

Before making routine measurements of the amount of auxin which can be diffused from sections pretreated with auxin, it is necessary to know the progress curve for the unloading of auxin from the sections. Experiments were carried out under numerous types of circumstances to determine this, and an example is presented in Table II. In this case, donor blocks were applied over a wide range of times (2 to 18 hr); the donors were removed, the sections were placed with their physiological base on plain agar, and the receptor discs were replaced every hour for 4 hr. In each instance, over 93% of the diffusible auxin had been exported from the sections at the end of 2 hr. For this reason, then, 2-hr unloading periods were utilized as standard procedure.

In some of the earliest work on auxin transport, van der Weij (26) noted that when a donor supply was continuously present, the amount of auxin accumulating in a receptor block increased in an approximately linear fashion with time; this suggests that one might be able to measure transport as a steady state process. In order to look more precisely at the dynamics of the delivery of auxin to receptors, sunflower sections of 2-, 4-, or 8-mm length were provided with continuous IAA-^{14}C, and the amounts of auxin arriving in receptor blocks were measured by replacing the receptors each hour. The results shown in Figure 1 indicate that the auxin delivered out of 2-mm sections reaches approximately a steady state in less than 2 hr, and the longer sections reach a steady state at about 3 hr. Until a steady state of delivery is achieved, the arrival curve (Fig. 1A) may be slightly concave (*cf.* 3, 11, 13, 24).

Another type of experiment which may be useful in measuring the capacity of the tissue to deliver auxin involves supplying the donor source of auxin for various intervals of time and determining the amount of auxin which can be subsequently diffused from the tissue, as in Table II. Using a uniform time period for the diffusion of auxin from the tissue, the data in Figure 2 reveal that the amount of diffusible auxin is surprisingly uniform even when enormously different amounts of auxin have been taken up by the tissue. Whereas the amount of auxin in the tissue increases linearly with time, the amount diffusible from the tissue (after the donor has been removed) does not, becoming fairly constant after the completion of about 3 hr of uptake. The steady rate of delivery of auxin (Fig. 1) and the rather constant amount of auxin diffusible from previously loaded sections (Fig. 2) are consistent with the concept of a restricted pool of transportable auxin or auxin transport sites.

The procedure of filling the tissue with a pulse of auxin and then subsequently allowing unloading may give an estimate of the transportable pool or transport sites, and by delaying the pulse for various times it should be possible to detect changes in these categories with time. Toward this end we cut 6-mm sunflower sections and held them on plain agar for periods up to 25 hr after which they were recut, removing 2 mm from the basal end; radioactive donor blocks were then applied for 3 hr, after which the amount of diffusible auxin was determined over a 2-hr unloading time. Young and older plants (9 and 16 days, respectively) were compared simultaneously. The results shown in Figure 3 reveal a gradual loss of transport effectiveness after 3 hr. In both older and younger plant tissues, there was a 50% decline in the ability to transport auxin in slightly more than 10 hr. The amount of auxin entering the tissues was essentially unchanging for the various aging periods, being 14,000 to 16,000 cpm for the older tissues and 15,000 to 20,000 for the younger; the decline in transport evident in Figure 3 is not related to a decline in the supply of auxin in the tissue.

Another type of information about the transportable pool might be obtained by filling the sites in the tissue with a pulse of auxin and then following the changes in diffusible auxin after various periods of time. Toward this end we cut 4-mm sections and placed them inverted on donor blocks for a 2-hr uptake period; the sections were then transferred to plain agar, and receptor blocks were placed on the basal ends after the passage of various periods of time. Diffusion of auxin from the tissue was allowed for 2 hr in each instance, and simultaneous experiments were done with younger and older sunflower plant material (8 and 15 days, respectively). The results shown in Figure 4 are quite

Table I. *Effect of Recutting on Transport of Auxin by Sunflower Hypocotyl Sections*

Sections cut 4, 5, or 6 mm were aged for 2 or 24 hr and inverted on plain agar or agar containing 10^{-5} M IAA. They were recut to 4 mm and a donor block (10^{-5} M IAA-^{14}C) was applied to the apical end for 2 hr, followed by a receptor block at the basal end for next 2 hr.

Treatment	Diffusible Auxin	Tissue Auxin
	cpm	*cpm*
Aged 2 hr	3,056 ± 98	6,060
Aged 24 hr		
No recut	340 ± 54	22,000
Apex recut	270 ± 50	9,200
Base recut	1,633 ± 125	19,300
Apex and base recut	1,269 ± 33	8,000
Unlabeled IAA 24 hr		
No recut	60 ± 10	13,000
Apex recut	186 ± 17	8,450
Base recut	2,244 ± 194	11,100
Apex and base recut	2,559 ± 57	6,260

Table II. *Time Requirement for Unloading Auxin from Hypocotyl Sections*

Six-millimeter sections were placed apical end down on donor blocks for 2 to 18 hr; they were then removed, the base was recut to remove 1 mm, and they were placed upright on receiver blocks which were renewed every hour.

Uptake		Diffusible Auxin				
Duration	Total	1st hr	2nd hr	3rd hr	4th hr	Total
hr	*cpm*			*cpm*		
2	6,150 ± 184	1,920 ± 42	345 ± 16	90 ± 3	28 ± 3	2,383 ± 59
4	13,420 ± 422	1,734 ± 17	516 ± 29	93 ± 8	33 ± 8	2,376 ± 30
6	15,740 ± 470	2,328 ± 29	204 ± 14	47 ± 1	33 ± 2	2,612 ± 28
12	23,600 ± 967	1,531 ± 104	965 ± 41	131 ± 18	58 ± 4	2,685 ± 90
18	35,860 ± 1,846	1,467 ± 143	330 ± 44	86 ± 16	38 ± 13	1,921 ± 125

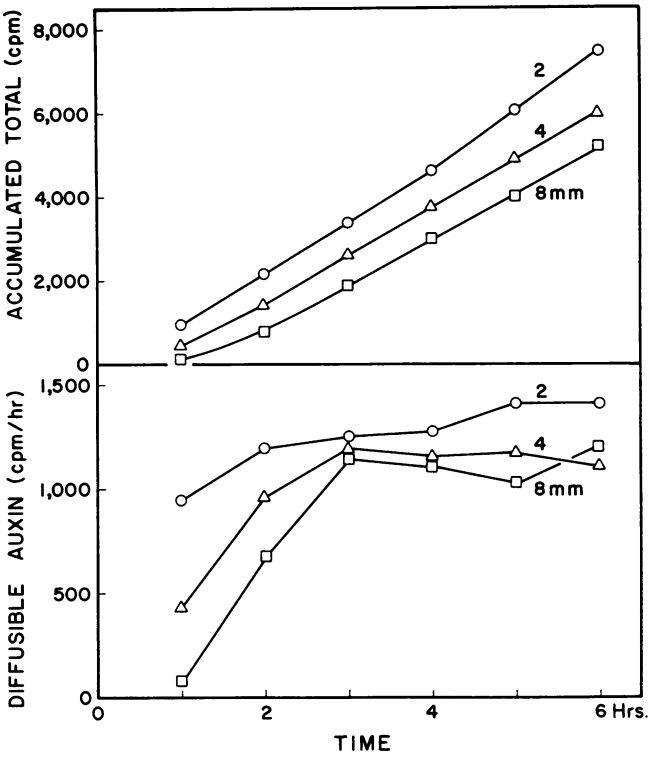

FIG. 1. The transport of IAA-^{14}C through sunflower stem sections 2, 4, or 8 mm long; data are plotted as the total radioactivity accumulated in the receptor blocks (above) and as the amount arriving in the receptors per hour (below). Donors were continuously present.

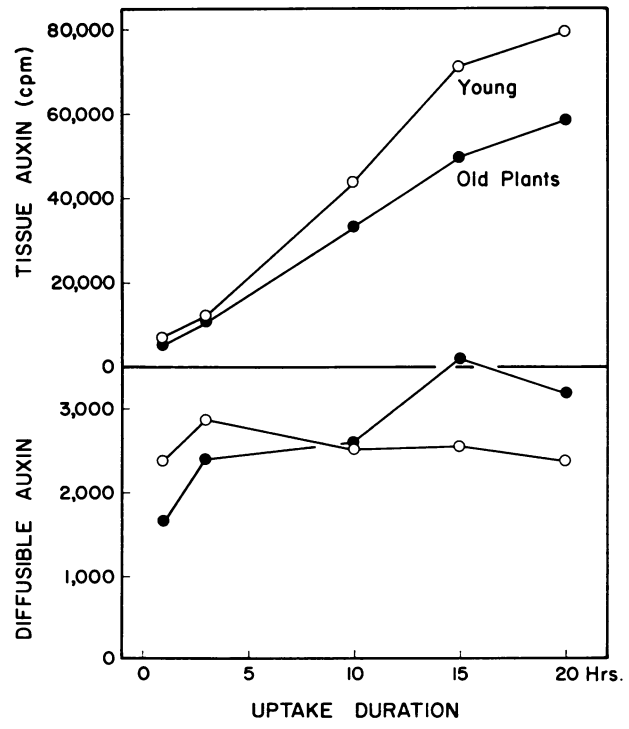

FIG. 2. Auxin transport after donor applications of 1 to 20 hr time, showing the radioactivity in the tissue (above) or in the receptor block (below) after being recut and placed on a receptor block for a standard 2-hr drain period. Young and old plants were 9 and 16 days old, respectively.

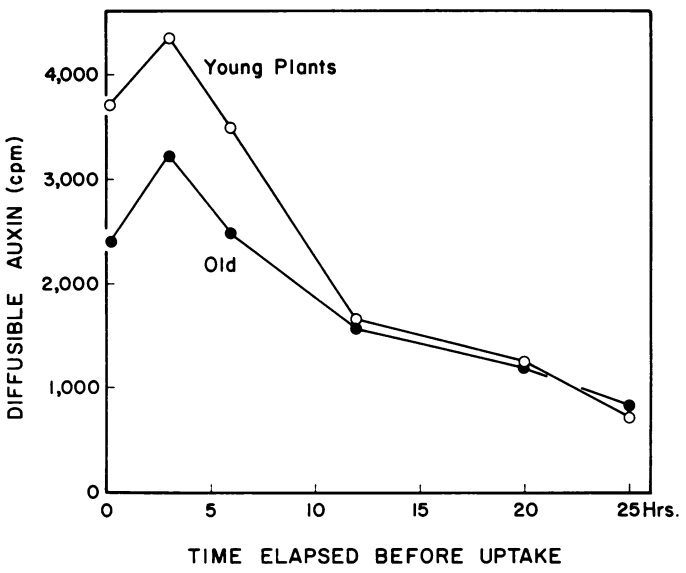

FIG. 3. Changes in the ability of sunflower sections to transport auxin with 0 to 25 hr of aging after cutting the sections. After the aging period indicated, sections were recut and donor blocks were applied for 3 hr, after which receptor blocks were applied for 2 hr. Young and old seedlings were 9 and 16 days old, respectively.

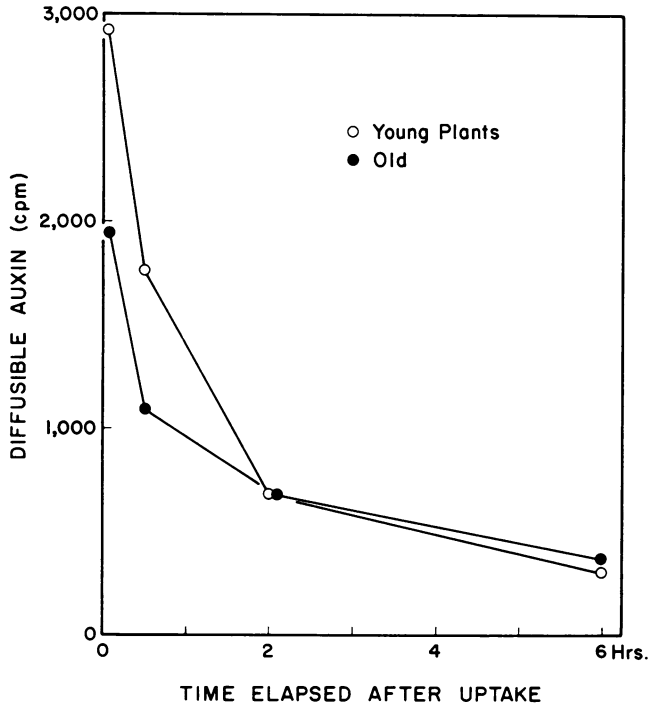

FIG. 4. Changes in the transportability of a pulse of auxin. Donor block was applied for 2 hr, and receptor blocks were then applied for 2 hr after 0 to 6 hr. Young and old plants were 8 and 15 days old, respectively. Sections were not recut.

different from those in Figure 3, the decline in diffusible auxin being markedly more rapid both in the younger and older tissue. Diffusible auxin fell to about half of its initial value after 1 hr. We suggest that this may indicate a half-life of the transport pool of about 1 hr. The decline of this pool is an order of magnitude more rapid than that of the over-all transport effectiveness as deduced from Figure 3.

The possibility that auxin which follows labeled auxin in the

transport system might alter the transport effectiveness was tested by Goldsmith and Thimann (7). In two experiments they applied a pulse of radioactive IAA followed by unlabeled IAA; they found no detectable effect of the cold "chase" auxin. We have tested this possibility in several different ways, including the experiment shown in Figure 5. In this case, sunflower sections were given a 2-hr pulse of IAA-^{14}C from a donor block, after which they were placed with the apical end on plain agar, on agar containing unlabeled IAA, or on agar containing IAA-^{14}C, all at 10^{-5} M. Receptor discs were applied for each of four 1-hr intervals. The results indicate that there was no alteration of the amount of auxin transport by the "chase" of unlabeled auxin.

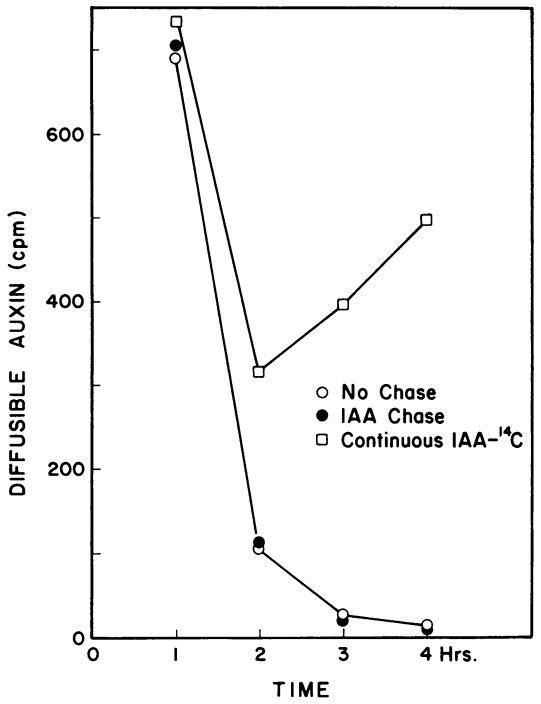

FIG. 5. The effect of a chase of unlabeled auxin following a 2-hr uptake of IAA-^{14}C. Receptor blocks were changed each hour, and the radioactivity in each was recorded.

A complementary experiment can be done, testing the possible effects of previously loaded auxin on the subsequent transport. Results of such a test are presented in Figure 6, where it is evident that loading the sections with unlabeled IAA for 2 hr resulted in 40% inhibition of subsequent transport of labeled IAA. With the passage of time, during which the sections were held between plain agar blocks, the inhibitory effect was 50% alleviated after about 1 hr.

Since it has been previously reported that pretreatment of auxin-depleted tissues with auxin enhanced the subsequent transport effectiveness (10, 17, 22), it appeared that the inhibition by auxin pretreatment might be associated with the freshly cut condition in which the transport system was initially operating efficiently. In several types of tests, we have compared the effects of a cold auxin pretreatment on the transport of a subsequent pulse of labeled auxin at various intervals of time after cutting, and in every instance the data have shown that the pretreatment depressed auxin transport in freshly cut sunflower sections and promoted transport in sections that had been aged. A representative experiment is reported in Figure 7. If the stem sections are aged for 2 hr after cutting before applying the 2-hr pulse of labeled auxin, an increase in transport effectiveness is observed (as in Fig. 3), and then with further periods of aging there is a decline in transport. Pretreatment of the sections with cold auxin resulted in an inhibition of transport at 2 hr after cutting, no effect at 12 hr, and a pronounced promotion of transport at 24 hr after cutting. We suggest that the pretreatment with cold unlabeled auxin competitively inhibits transport in freshly cut sections where endogenous auxin is abundant and transport sites tend to be already filled. Pretreatment of aged sections promotes transport, apparently through a different type of action, perhaps through a stimulation of metabolic activities. Such a stimulatory effect by auxin is evident also in Table I in recut, aged sections; likewise the rise in transport in sections from older plants over a 15-hr period of auxin supply seen in Figure 2 may reflect the same sort of auxin stimulation.

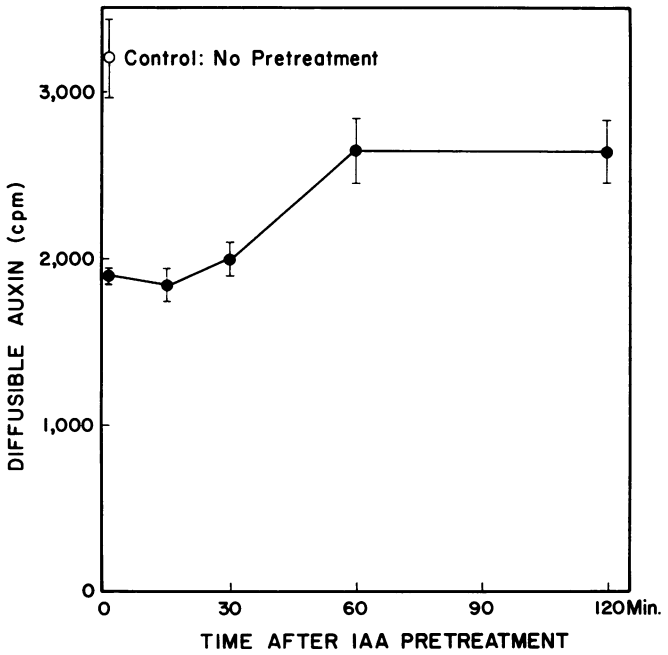

FIG. 6. The inhibition of transport of IAA-^{14}C by previously applied IAA. Donor blocks containing unlabeled IAA (10^{-5} M) were applied for 2 hr, and sections were placed between blank agar blocks for the time indicated on the abscissa, followed by 2 hr with the radioactive donor, and then 2 hr on receptor blocks.

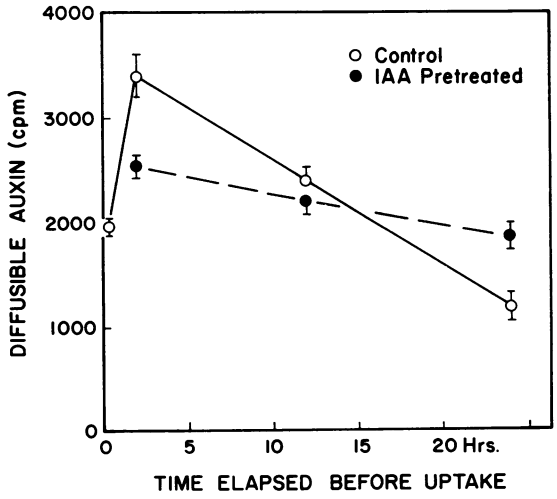

FIG. 7. The effects of pretreatment with unlabeled auxin as a function of aging before transport of IAA-^{14}C. Sections 5 mm were given unlabeled auxin donors for time indicated on abscissa, after which the base was recut to 4 mm and radioactive donors were applied (2 hr) followed by receptor blocks for 2 hr.

DISCUSSION

The experiments reported here provide evidence for a steady state of auxin transport in sunflower stems, a deterioration of the capacity of the tissue to transport auxin with aging, a deterioration of the availability of a pulse of auxin for transport, and interactions between sequentially applied pulses of labeled and unlabeled auxin. Collectively, we suggest that these data are interpretable around the concept of a limited pool of transportable auxin, and a limited supply of transport sites.

The steady state of auxin transport, which is expressed in the nearly linear arrival curves of transport (Fig. 1) and the rather comparable amounts of transportable auxin over a wide range of tissue contents (Fig. 2), is obviously consistent with the interpretation of a limited pool of transportable auxin and auxin sites. The rather slow decline in the capacity of the tissue to transport auxin with aging (Fig. 3) indicates either a decline in the numbers of available transport sites or an increasing ability of the tissue to destroy or immobilize auxin. The rapid decline in transportability of a pulse of auxin (Fig. 4) suggests that there is a rapid turnover either of the auxin in the transportable pool, or of transport sites, or both. As in the case of the decline of capacity of the tissue, destruction or immobilization of auxin may contribute or even account for this decline; this possibility does not detract, however, from the inference that there is a rather rapid turnover of the transportable auxin pool. The experiments on competitions between pulses of labeled and unlabeled auxin (Figs. 5, 6, and 7) are readily interpretable as evidences of competition for a limited number of transport sites. The inhibition of transport of labeled auxin by a previous application of unlabeled auxin strongly suggests a filling of the available pool, and the persistence of this inhibition over a time period equivalent to the persistence of auxin in the transportable state is consistent with this interpretation.

Some of our results reported here may reflect the immobilization and destruction of auxin. There are numerous instances in the literature in which auxin has been found to be available to extraction but not capable of diffusion out of the tissue via the transport system (23). The diversion of auxin into nontransportable forms was recognized by Goldsmith and Thimann (7) and subsequently described by others (5, 9, 25, 27). This diversion of auxin may or may not involve a chemical transformation of the auxin (27). Turning to the experiments reported here, it is highly unlikely that auxin destruction or immobilization would account for the steady state of auxin transport (Figs. 1 and 2). However, it might well contribute to the decline in the capacity of the tissue to transport (Fig. 3) and the decline in transportability of a pulse of auxin (Fig. 4). Whatever its fate, auxin appears to become rapidly inaccessible for transport after entry into the tissues used in these experiments, suggesting a rapid turnover of the transportable auxin pool. The uniformity of the steady state of auxin delivery, regardless of the amount of tissue traversed (Fig. 1), indicates that the auxin which is being actively transported (from transport site to transport site, according to the interpretation we are suggesting) is relatively immune to such immobilization or inactivation reactions, as has been suggested long ago by Briggs *et al.* (1).

The central hypothetical deduction that we suggest from these data is that limitations on the pool of transportable auxin and available transport sites may establish the capacity of the sunflower tissue to transport auxin. The availability of sites may be presumed to be a product of the rate of formation of new sites (generation or regeneration) minus the rate of site deterioration. The data in Figure 4 suggest an outer limit for the half-life of transportable auxin as 1 hr. The data in Figure 3 suggest a limit for the decline in total site availability as a 50% decline in 10 hr.

The existence of a declining gradient of auxin transport down a stem or coleoptile is well known (12, 17, 23). In our experiments, sections taken from older stems can deliver previously loaded auxin at about the same maximal rate as sections from younger stems (Fig. 2), and the apparent half-life of the transport pool is 1 hr in both younger and older stems (Fig. 4), but the lesser transport of auxin over short periods of time (Figs. 2, 3, and 4) suggests that the generation of transport sites may be lower in the older tissues. We suggest, then, that the auxin transport gradient from the youngest apical tissues to the older basal tissues may be a consequence of a declining rate of generation of auxin transport sites.

Previous workers have noted that when auxin transport experiments are continued for longer periods of time (4–8 hr), there is a deterioration of the ability of the tissue to deliver the auxin (7, 20). From Table I it is evident that the basal cut surface becomes limiting with time, and this may be the reason for the decline if the sections are not recut.

It has been suggested that the site of auxin transport (or secretion) from cells may be at the plasmalemma (15, 16). Pardee (19) has developed the concept that transport of solutes across membranes may involve specific protein entities in the membrane—transport proteins. It is attractive to speculate that the generation of transport sites for auxin might involve the synthesis or alteration of protein constituents in the plasmalemma. The ability of auxin to move out of cells in any direction, even in tissues which are quite polar in their transport of auxin (16), suggests that such transport sites may exist on all sides of the cell. Polarity of transport may then be a consequence of a preferential activation of such sites at the lower end of each cell, and tropistic responses with associated lateral transport of auxin (4, 8) may involve a shift in the pattern of activation of auxin transport sites in the lateral walls of the cells.

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