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Action of (2-chloroethyl) trimethylammonium chloride, 2,4-dichlorobenzyl-tributylphosphonium chloride, and N-dimenthyl-amino-succinamic acid on IAA and coumarin induced growth of sunflower hypocotyl sections *

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I. INTRODUCTION

The opinions bearing on the mode of action of growth retardants (cf. reviews: Cathey 1964; Knypl 1966a) may be classified into three groups: (1) growth retardants act as inhibitors of GA biosynthesis (Ninnemann et al. 1964; Harada and Lang 1965; Dennis et al. 1965; Baldev et al. 1965; Zeevaart and Osborne 1965); (2) growth retardants are antagonistic to the auxin system in plants (Kuraishi and Muir 1964; Knypl 1964a, b; Cleland 1965); and (3) neither GA nor IAA prevent or reduce the inhibitory effect of growth retardants (Kuraishi and Muir 1963; Sachs and Wohlers 1964; Murashige 1965): their mode of action remains, therefore, obscure in these instances (e.g. radish leaf discs, in vitro cultivated callus tissues).

Knypl has found that the retarding action of CCC on the growth of sunflower hypocotyl sections was reversed by coumarin (Knypl 1964a) and IAA (Knypl 1964b). CCC did not affect the GA-stimulated growth of the first leaf of maize (Knypl 1964b). The aim of the experiments reported here was to test whether or not the natural regulators: GA, kinetin, IAA and coumarin will be capable of overcoming the retarding effect of CCC on the growth of sunflower hypocotyl sections, and to compare the effects brought about by CCC with those induced by other growth retardants, Phosfon D and B995.

The following abbreviations will be used: B995—N—dimethylamino-succinamic acid; CCC—(2-chloroethyl) trimethylammonium chloride; Phosfon D—2,4-dichlorobenzyl-tributylphosphonium chloride; GA—gibberellic acid; IAA—indolyl-3-acetic acid; AMC—actinomycin C_1; COU—coumarin; KIN—kinetin.

II. MATERIAL AND METHOD

Experiments were carried out on hypocotyl segments taken from 6-7-days-old sunflower plants (*Helianthus annuus* L. var. Pastewny) grown in the dark according to a technique described previously (Knypl 1964a).

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Hypocotyl segments 10-mm, dissected out about 5 mm below the cotyledonar node of the 7—10 cm long sunflower seedlings, were floated on distilled water for 2 hours, washed with tap water, divided into groups of eight, weighed on a torsion balance and transferred into 5,5-cm Petri dishes previously filled with 10 ml of a solution of the substance(s) under examination. Covered dishes were incubated under continuous artificial illumination (Knypl 1964a) at 24.5°C or at 26—27°C. Tests with actinomycin C_1 were carried out in the dark. Growth of the sections was measured gravimetrically.

Each type of test was carried out in duplicate and replicated 4 times.

III. RESULTS

A. (2-Chloroethyl) trimethylammonium chloride

Preliminary tests have revealed that CCC in solutions more diluted than 5×10^{-3} M did not practically affect the longitudinal extension of sunflower hypocotyl sections. Marked reduction in growth, up to 50 per cent of the control rate, was produced by 10^{-2} M of CCC. At the concentration of 5×10^{-2} M CCC dramatically arrested growth; nevertheless, it did not induce any visible intoxication symptoms (Knypl 1964a).

The growth rate of sunflower hypocotyl sections incubated in distilled water is not constant in time, and continuosly decreases after the initial 10—12 hours. For this reason the retarding effect of CCC (10^{-2} M) was the most evident in relatively short-timed tests, lasting up to 10—12 hours. After 24 hours the fresh weight of the sample treated with CCC was nearly the same as in the control, because in the former sample growth — though slower — was more prolonged in time (cf. Knypl 1964a). Such a result indicates that CCC actually does not inhibit but retards growth.

The data plotted in Fig. 1 show that CCC at the concentration of 8.2×10^{-3}M markedly retarded the longitudinal extension of sunflower hypocotyl section, whereas IAA (10^{-5} M) and coumarin (200 p.p.m.) stimulated it. Coumarin completely reversed the symptoms of CCC action during the initial 10—11 hours: the growth rate of the (CCC+Coumarin)-treated sample was nearly identical with that of the water-treated control. IAA was more effective in this respect, because the growth rate in the (CCC+IAA)-treated sample was equal to that in the sample subjected to coumarin, that is it was about 2—2.5 times higher as compared with that in the control. It may be concluded, therefore, that coumarin and IAA reverse the retarding effect of CCC or, in other words, CCC abolishes the IAA and coumarin induced growth.

As reported previously (Knypl 1964a) and as seen from the data in Fig. 1, the growth-promoting effects of IAA and coumarin are not additive. On the contrary, in these conditions coumarin markedly decreases the net IAA-induced growth, and the growth rate of the (IAA+Coumarin)-treated section continuously decreases with prolongation of the time of incubation. This indicates that IAA- and coumarin-

induced growth phenomena are different in nature, and agrees with the results of other experiments which revealed that coumarin-induced growth, in comparison with that induced by IAA, was more inhibited by uracil analogues and was more resistant to the action of AMC, puromycin and chloramphenicol (Knypl 1965). It is not astonishing, therefore, that the growth rate in the (CCC+IAA+Coumarin)-

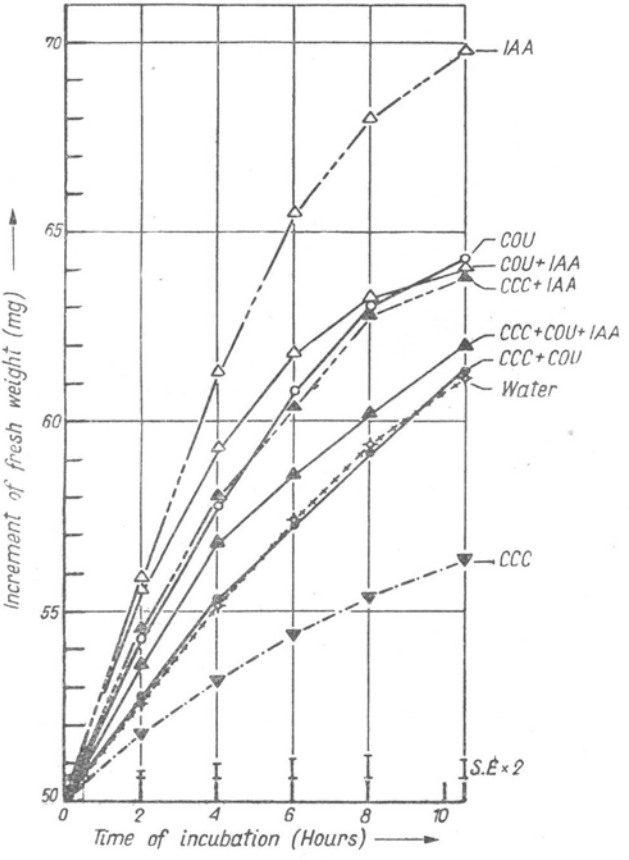

Fig. 1. IAA and coumarin-induced reversal of the retarding effect of CCC on the growth of sunflower hypocotyl sections. IAA, 10^{-5} M; COU, 200 p.p.m.; CCC, 8.2×10^{-3} M. The final concentration of the compounds in mixtures are the same as in the case of single solutions. S.E., standard error. After 30 hours of incubation at 26—27° C in the light the sections weighed (mgm.): IAA—73.69; COU — 62.60; COU+IAA, — 63.45; CCC+IAA, — 65.77; CCC+COU+IAA—65.41; CCC+COU—65.41; WATER—65.00; CCC—61.17. Initial weight of one section=50.0 mgm.

treated sample was somewhat increased in comparison to that in the (CCC+Coumarin)-treated series, but was markedly lowered in comparison with that in the sample incubated in (CCC+IAA).

It had been reported that growth effects brought about by coumarin in sunflower hypocotyl sections were highly dependent on temperature (Knypl 1964a). The temperature of 25—26°C appeared to be a threshold value. Coumarine in a con-

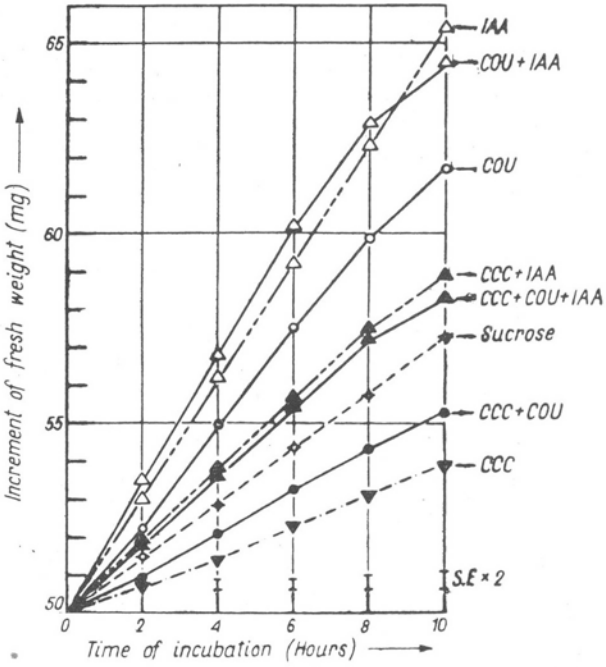

Fig. 2. Effect of sucrose and lower temperature on the IAA-and coumarin-induced reversal of the retarding effect of CCC on growth of sunflower hypocotyl sections. All the substances tested were dissolved in 2.0 per cent sucrose; concentrations as in Fig. 1. After 30 hours at 24.0—24.5° C the sections weighed (mgm.): IAA — 73.04; COU+IAA — 69.42; COU — 69.60; CCC+IAA — 64.20; CCC+COU+IAA — 62.26; SUCROSE — 63.00; CCC+COU — 61.70; CCC — 59.35.

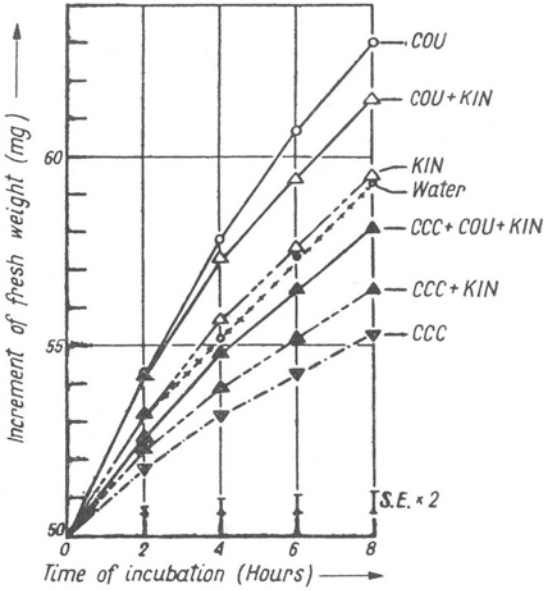

Fig. 3. Effect of kinetin on the CCC-dependent retardation of growth of sunflower hypocotyl sections. KIN, 10^{-5} M. Other details as in Fig. 1; growth rate of the (CCC+COU) affected section is presented in Fig. 1.

centration of 200 p.p.m. never induced visible symptoms of poisoning of the sections incubated for 30 hours at 20—25°C. In contrast, if the temperature of the environment exceeded 26°C, the coumarin-treated sections exhibited visible symptoms of intoxication, i.e. browning and flexibility, sometimes as early as after 12 hours. Sucrose was found to be a protective agent in this case.

The data plotted in Fig. 1 have been gathered in experiments carried out at 26—27°C. Analysis of fresh weight of the sections incubated for 30 hours reveals that the coumatin-affected section weighed 62.5 mgm, while after 11 hours it weighed 64.2 mgm, i.e. about 2 mgm more. Fresh matter of the (CCC+Coumarin)-treated section was 65.4 mgm as compared with 65.0 mgm in the water-treated control and 61.4 mgm in the section subjected only to CCC. These data indicate that CCC reverses the visible symptoms of intoxication due to coumarin.

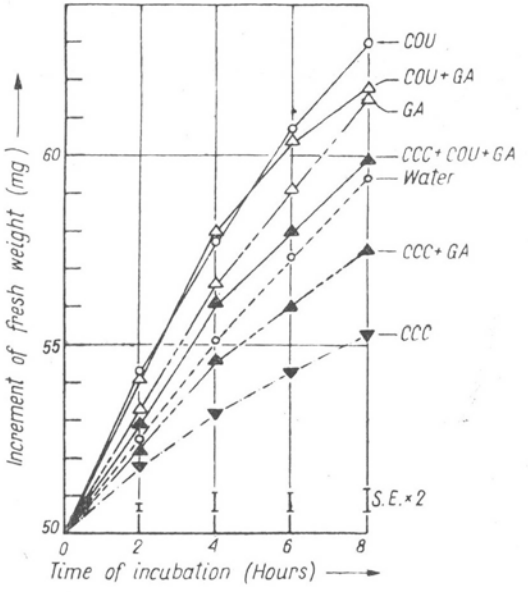

Fig. 4. Effect of gibberellic acid on the CCC-dependent retardation of growth of sunflower hypocotyl sections. GA, 10^{-5} M. Other details as in Fig. 1 and Fig. 3.

The next series of analyses were carried out at 24.5°C with application of the growth regulators dissolved in 2.0 per cent sucrose. In these conditions coumarin did not produce any symptoms of poisoning. As seen from the data plotted in Fig. 2, the efficiency of coumarin in reducing the CCC-dependent retardation of growth decreased to about one half in this case. (In other series of analyses, carried out in 1965 and 1966, such a decrease has been not recorded). The degree of interference of coumarin with IAA also decreased. Moreover, the samples treated with a mixture of (Coumarin+IAA) during the initial 8 hours grew more intensively than the sections immersed in IAA alone. Despite this initial synergism between coumarin and the auxin, coumarin did not exert any positive effect on the growth of the (CCC+IAA)-affected sections (Fig. 2).

Kinetin used in the optimal concentration of 10^{-6} M, insignificantly accelerating growth in the control, decreased to some degree the retarding effect of CCC, and — especially after 4 hrs. of action — the coumarin-induced growth (Fig. 3). Consequently, the growth rate of the (CCC+Coumarin+KIN)-treated sample was lowered as compared with that of the (CCC+Coumarin)-treated sample (cf. Fig. 1 and Fig. 3). Therefore, the activities of kinetin and coumarin as agents reversing the CCC-dependent retardation of growth are not additive.

Gibberellic acid, like kinetin, reversed also rather slightly the CCC-dependent retardation of growth. Nevertheless, in contrast to kinetin, GA enhanced growth rate in the series treated with a combination of (CCC+Coumarin+GA) in comparison with that in the series incubated in (CCC+Coumarin) (cf. Fig. 4 and Fig. 1). GA exerted no positive effect on growth of the coumarin-affected sample.

Table 1

Effect of actinomycin C_1 on growth of the CCC-affected sunflower hypocotyl sections

Treatment ***	Increment of fresh matter (mgm)				
	Time of incubation (Hours)				
	0	4	8	12	20
0 (only 2 % sucrose)	50.0	53.52	56.55	59.61	64.57
CCC, 8.2×10^{-3} M	50.0	51.84	54.24	56.41	60.08
AMC, 7.0 µg/ml	50.0	52.95	55.60	57.40	60.80
CCC+AMC	50.0	52.15	54.14	56.23	59.00
AMC-4 hr.*+CCC	50.0	52.95**	55.80	58.06	60.55
AMC-8 hr.*+CCC	50.0	52.95	55.60**	57.40	60.47

* AMC-4 hr. and AMC-8 hr.: The sections were preincubated in AMC for 4 and 8 hours, respectively, and thereafter transferred into a CCC solution without AMC.

** Moment of transfer of the sections from AMC to CCC.

*** All solutions contained 2.0 per cent sucrose; in the case of (CCC+AMC) mixture, as in the case of single solutions, the final concentration of AMC was 7.0 µg/ml and CCC 8.2×10^{-3} M. Other details as in Fig. 5.

It has been suggested that IAA and coumarin-induced growth of sunflower hypocotyl sections may be dependent on the induced and/or increased synthesis of ribonucleic acids regulating the synthesis of specific enzymes, i.e. messenger RNAs (Knypl 1965). Experiments carried out with application of actinomycin C_1, known as a specific inhibitor of the DNA dependent RNA synthesis, have revealed that this inhibitor did not increase the retarding effect of CCC on growth (Table 1). After 4 hours of action, AMC (7.0 µg/ml) even slightly decreased the effect of CCC, since CCC- and (CCC+AMC)-affected sections weighed 51.8 and 52.1 mgm, respectively (initial weight 50.0 mgm). This tendency was more pronounced when CCC was applied 4 hours after AMC. The section pretreated for 4 hours with AMC weighed 52.9 mgm; during the subsequent 4 hrs of incubation in CCC the fresh matter increased to 55.8 mgm, that is 2.8 mgm net. On this basis it may be concluded that (1) in the CCC-affected sunflower hypocotyl sections the rate of synthesis of RNA is markedly reduced, and (2) that CCC can retard the growth

by inducing the synthesis of some compound(s) (protein or RNA), which reduces the rate of normal growth by interfering with the synthesis of messenger ribonucleic acids.

The data of Fig. 5 show that AMC, applied simultaneously with IAA and CCC at the beginning of the test, drastically reduced the ability of IAA to reverse the

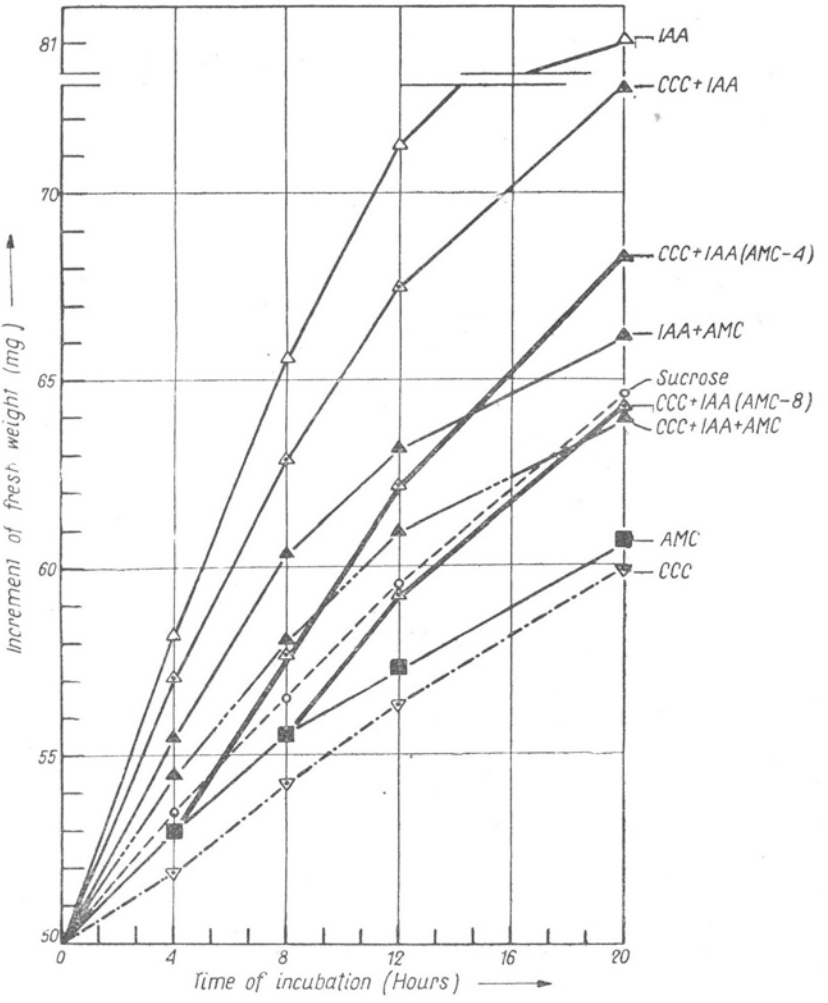

Fig. 5. Effect of actinomycin C_1 on growth of sunflower hypocotyl sections affected with IAA and CCC. Final concentrations: IAA, 10^{-5} M; CCC, 8.2×10^{-3} M; AMC, 7.0μg/ml. Each solution of the substances under examination contained 2.0 per cent sucrose. Experiments were carried out in the dark at 24.0—24.5° C. (AMC-4) and (AMC-8) — sections preincubated in AMC (7.0μ g/ml) for 4 or 8 hours, respectively, and thereafter transferred into a mixture of (CCC and IAA) without AMC (thick lines). Growth in the series [IAA (AMC-4)] and [IAA (AMC-8)], not recorded graphically, was the same as in the series [CCC+IAA (AMC-4)] and [CCC+IAA (AMC-8)], respectively. Growth in series (CCC+AMC), [CCC (AMC-4)] and [CCC (AMC-8)] is recorded in Table 1.

retarding effect of CCC. If IAA and CCC were applied to sections previously pretreated for 4 or 8 hours with AMC, then CCC did not reduce significantly the stimulatory effect of the auxin on growth. That is, pretreatment with AMC markedly reduced the extent of response of the sections to IAA added thereafter, but CCC in this case did not exert any effect on the following growth. After pretreatment with AMC, the subsequent growth in the series transferred to IAA was nearly the

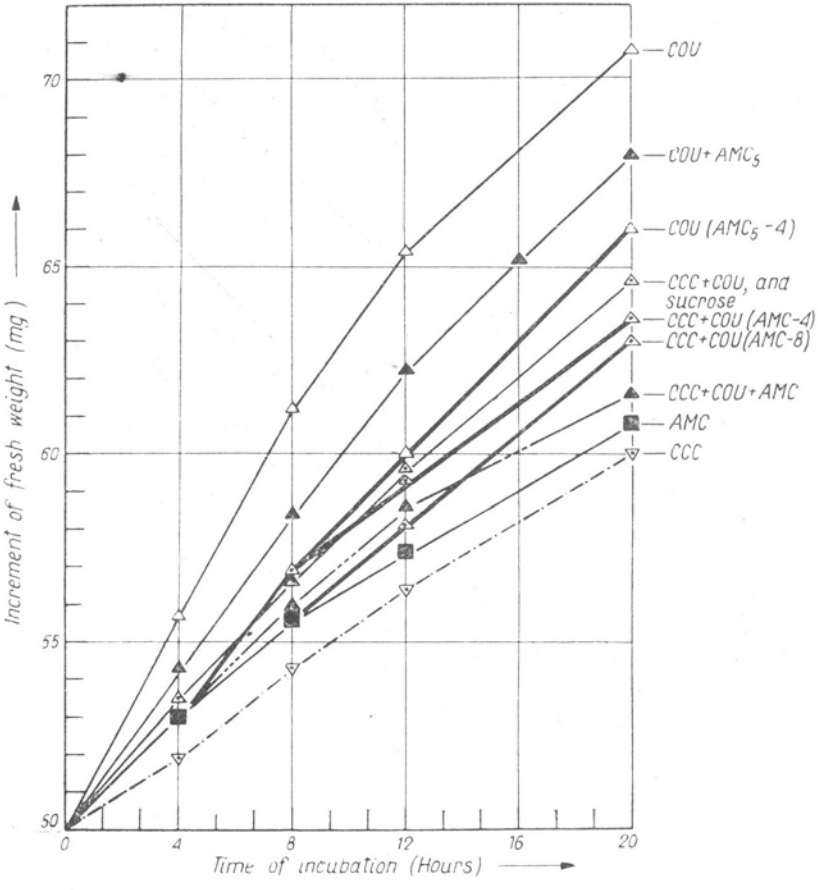

Fig. 6. Effect of actinomycin C_1 on growth of sunflower hypocotyl sections affected with coumarin and CCC. COU, coumarin 200 p.p.m.; AMC_5 , actinomycin 5.0μg/ml; AMC, actinomycin 7.0 μ g/ml. Other details as in Fig. 5; instead of IAA read COU.

same as in the series transferred to a combination of (CCC+IAA). Similar results have been noted also in other series of analyses, where AMC was applied in a concentration of 5,0 μg/ml (unpublished data).

These data indicate that IAA-induced reversal of the CCC retarding effect on growth is completely dependent on the IAA-induced and/or accelerated synthesis of RNA(s) in this system.

Actinomycin decreased also the growth in the samples treated with coumarin and CCC (Fig. 6). However, in contrast to the IAA-affected samples, the growth rate of the samples pretreated for 4 hours in AMC and thereafter transferred to a mixture of (CCC+Coumarin) was significantly lower as compared with the growth rate of AMC-pretreated samples, transferred to coumarin alone. This indicates that coumarin presumably counteracts CCC on another way than does IAA.

B. Effects of phosfon D and B995 on IAA and coumarin-stimulated growth of sunflower hypocotyl sections

As seen from the data plotted in Fig. 7, phosfon D in the concentration of 10 p.p.m. slightly stimulated growth of sunflower. In spite of this stimulation it markedly reduced growth in IAA- and, particularly, coumarin-treated sections. After 8 hours

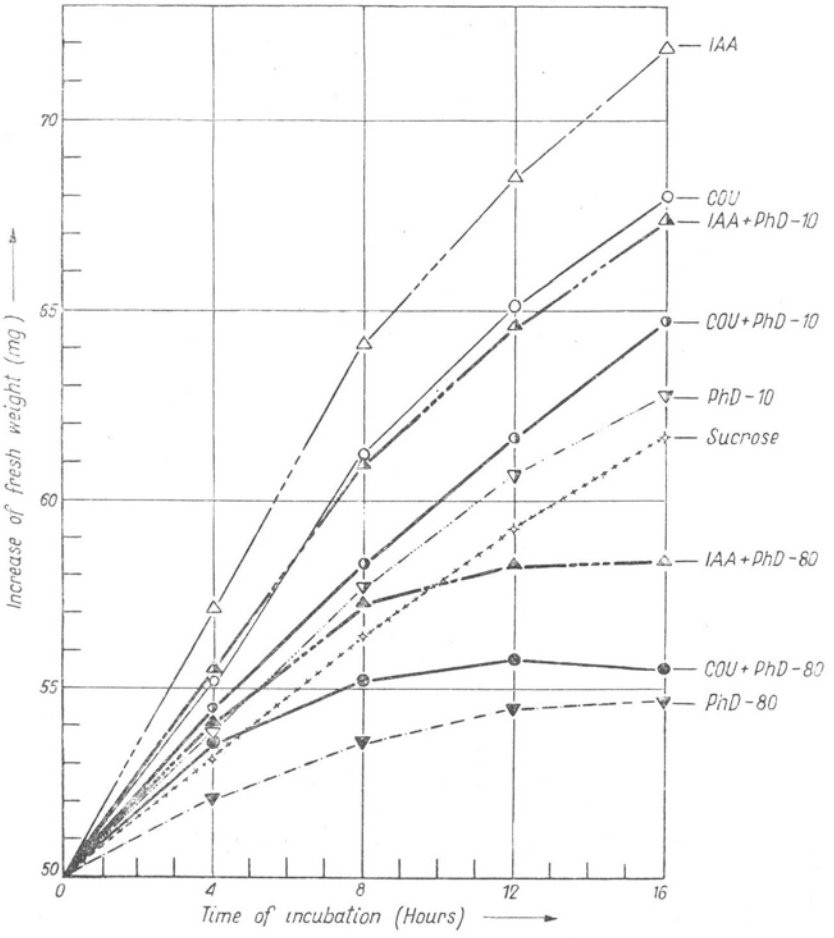

Fig. 7. Effect of phosfon D on growth of sunflower hypocotyl sections stimulated with IAA or coumarin. PhD-10 and PhD-80, phosfon D, 10 and 80 p.p.m., respectively; IAA, 10^{-5}M; COU, 200 p.p.m.; SUCROSE, 2.0 per cent. Incubation in the dark at 23° C.

of incubation there was practically no growth in the samples treated with combination of coumarin or IAA with phosfon D in the concentration of 80 p.p.m. It is worth noting that the sections incubated in the mixture (Coumarin+Phosfon D, 80 p.p.m.) became flexible and brown after 10 hours; this mixture is, therefore, very toxic for the plant material tested here.

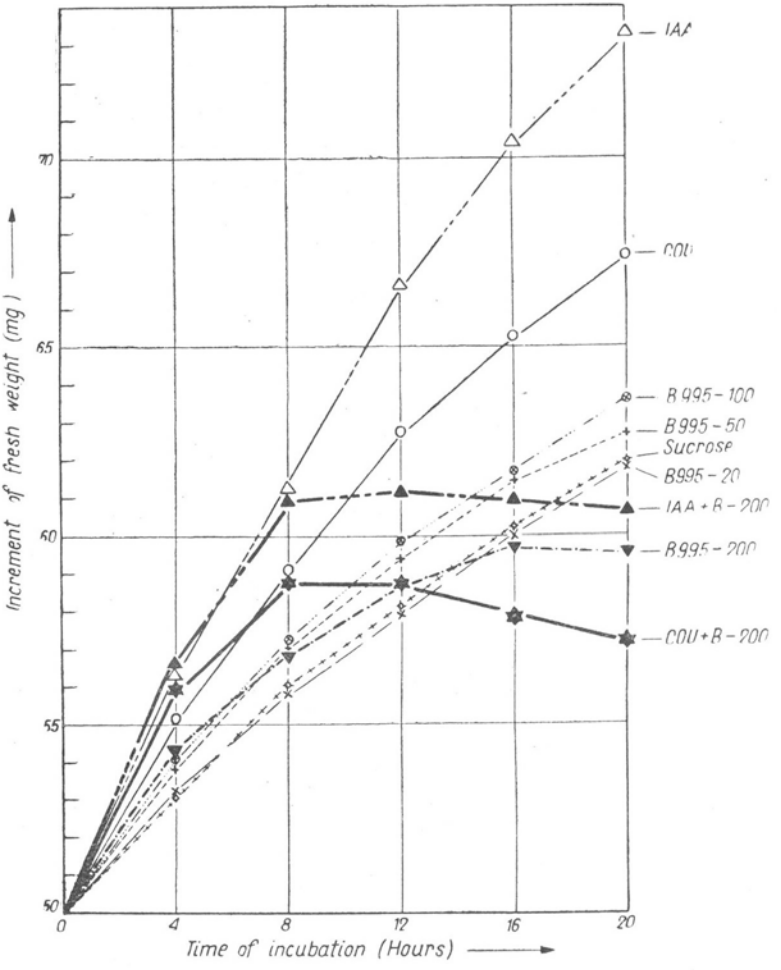

Fig. 8. Effect of B995 on growth of sunflower hypocotyl sections stimulated with IAA or coumarin. Concentrations: IAA, 10^{-5} M; COU, 200 p.p.m.; B995-20, B995-50, B995-100 and B995-200, N-dimethylamino-succinamic acid, 20, 50, 100 and 200 p.p.m., respectively. Each solution contained 2.0 per cent sucrose. Incubation was carried out in the light at 25° C.

B995 at concentrations of 50—100 p.p.m. promoted growth in sunflower hypocotyl sections (Fig. 8). More concentrated solutions of this growth retardant (200 p.p.m.) were without any effect on the longitudinal extension during the initial 8 hours of action, but completely arrested growth after 16 hours.

200 p.p.m. of B995 in a mixture with coumarin (200 p.p.m.) initially (4 hours) slightly enhanced growth. Nevertheless, after 8 hours of incubation growth in this series was completely stopped and the sections showed symptoms of poisoning as manifested by the loss of turgor and decrease in fresh matter. On the contrary, at a lower concentration of 100 p.p.m. B995 exerted positive effect on the growth of coumarin-affected sections. The data plotted in Fig. 9 show that the growth stimulating effects of coumarin and B995 (100 p.p.m.) are simply additive.

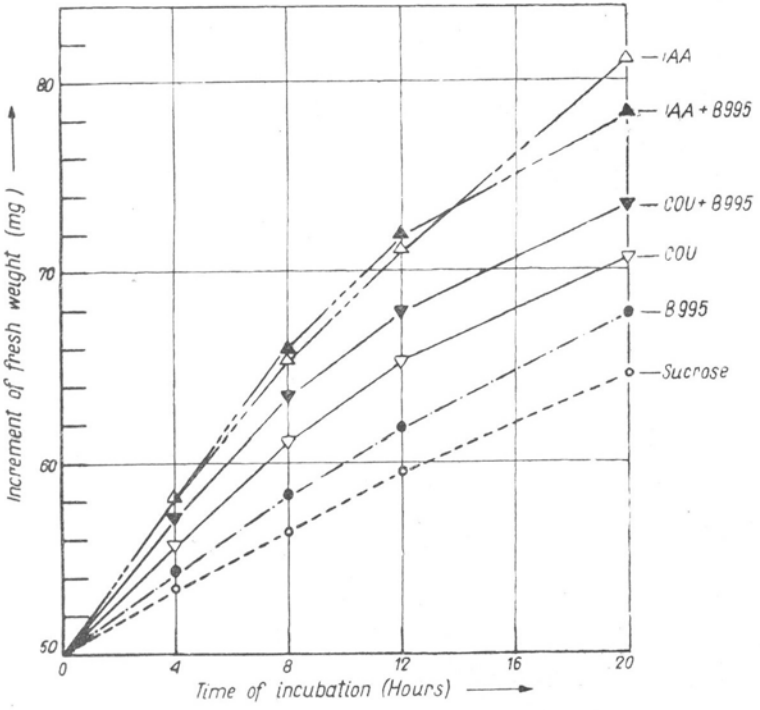

Fig. 9. Effect of B995 on growth of sunflower hypocotyl sections stimulated with IAA and coumarin. Final concentrations: IAA, 10^{-5} M; COU, 200 p.p.m.; B995, 100 p.p.m. Each solution contained 2.0 per cent sucrose. Incubation was carried out in light at 25°C.

B995 exerted no positive effect on growth in the IAA-treated samples. In the concentration of 100 p.p.m., B995 reduced elongation in the IAA-affected samples after 12 hours of incubation (Fig. 9), and at the concentration of 200 p.p.m. this growth retardant depressed growth in the series incubated longer than 8 hours (Fig. 8). However, in this case B995 did not produce as drastic toxic effects as in the case of coumarin-induced growth. These data (Fig. 8 and 9) substantiate the earlier inference that the mechanism of the growth-stimulating action of coumarin differs from that of IAA.

IV. DISCUSSION

The results of the experiments here described show clearly that the mechanism of the growth-retarding activity of CCC differs from that of phosfon D and B995.

As concerns CCC, it is clear that in the sunflower hypocotyl system which is relatively unsensitive to gibberellin, this growth retardant does not act as "anti-gibberellin" since the net increment of fresh matter in the series (CCC+GA) as compared with that in the proper control series treated with CCC alone is — within the limits of the standard deviation of the material — the same as the net increment of weight in the series treated with GA alone as compared with the control incubated in distilled water (cf. Fig. 4). It had been found that GA did not reverse the retarding effect of CCC and phosfon D on growth of *Avena* coleoptile sections (Kuraishi and Muir 1963) and *Avena* leaf sections (Cleland 1965), and that growth retardants did not interfere with the physiological activity of GA applied externally in the barley endosperm system (Paleg et al. 1965). It may be concluded, therefore, that CCC does not act at the site of GA action.

More striking results, in this study, have been produced by kinetin which in the concentration practically not stimulating growth in the control, significantly reduced the effects due to CCC (Fig. 3); at the same time kinetin reduced the growth-stimulating activity of coumarin. These tendencies, relatively little pronounced here, were markedly expressed in the case of germination of *Bassica* sp. seeds (Knypl 1966b). Kinetin (10^{-4} M — 5×10^{-4} M) significantly reduced the inhibitory effects of CCC, B995, phosfon D and coumarin on the germination of kale seeds. These data suggest that kinetin exerts its positive effect by means of regulation of RNA and protein synthesis. Results of the experiments carried out with application of AMC suggest that in the CCC-affected tissue the rate of RNA synthesis is lowered, and that IAA and coumarin antagonize the visible symptoms of the retardant action by accelerating the synthesis of RNA. It is possible that CCC induces the synthesis of some RNA or protein which act as the repressor of transcription of that piece of genetic information which is necessary for normal growth. This suggestion is supported by the fact that AMC-pretreated sections were less responsive to CCC in comparison with the control sections.

The data plotted in Figs. 1, 2, 5 and 6, show that at least part of the inhibitory action of CCC is due to an antagonism with the auxin metabolism in sunflower hypocotyl tissue, and that the primary mechanism of the reversing action of IAA is different from that of coumarin.

Since CCC itself retards the longitudinal extension of sunflower hypocotyl sections, it is not striking that it interacts with IAA and coumarin, and such a result could be anticipated. However, the fact that phosfon D in a concentration slightly stimulating growth (10 p.p.m.) reduces both the IAA and coumarin-induced growth could not be anticipated. This result (Fig. 7) seems to indicate that phosfon D directly antagonizes with IAA- and coumarin-stimulated metabolic pathways leading to the increased growth. The phosfon-blocked step in the case of both IAA and coumarin is probably the same.

Interpretation of the effects brought about by B995 is hardly possible on the basis of such scanty data as in Fig. 8 and 9. It has been found that B995, in contrast to CCC and AMO-1618, did not block the synthesis of gibberellin in *Fusarium moniliforme* (Ninnemann et al. 1964) and it was relatively ineffective in inhibiting the synthesis of (—)-kaurene from mevalonate in homogenates of endosperm and nucellus from seeds of *Echinocystis macrocarpa* Greene (Dennis et al. 1965). On the other hand, GA reversed the effect of this growth retardant on the geotropic response and shoot growth in Arizona cypress (Pharis et al. 1965) and in several angiosperms (Riddell et al. 1962). The "antigibberellinic" character of B995 and other growth retardants was not observed in tobacco callus culture (Murashige 1965). In contrast to other growth retardants, B995 did not affect the endogenous growth of *Avena* leaf sections (Cleland 1965).

Recently Reed has suggested that B995 in vivo may be converted to 1.1-dimethylhydrazine, known as a powerful inhibitor of diamine oxidase, and affects in this way the level of endogenous auxin in the plant by inhibiting the oxidation of tryptamine to indoleacetaldehyde (Reed et al. 1965). This suggestion seems to be incompatible with the data presented here, since B995 in a concentration slightly stimulating growth (100 p.p.m.) initially did not affect, and after 8 — 12 hours reduced, the IAA-stimulated longitudinal extension of the sections (Fig. 9). Rapid depression of growth in the IAA- and coumarin-stimulated sections, occurring after 8 hours of action of B995 (200 p.p.m., Fig. 8), and occurrence of a similar growth depression after 16 hrs. in the sample treated with B995 alone, suggest that this compund completely inhibits the synthesis of some metabolite(s) necessary for growth. B995 possibly acts here as the structural analogue of succinic acid.

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SUMMARY

IAA and coumarin reversed, and kinetin markedly reduced the retarding effect of CCC on the growth of sunflower hypocotyl sections; gibberellic acid was without any effect in this respect. The effects brought about by IAA and coumarin were not additive. On the contrary, these growth stimulators counteract each other if the temperature of incubation is higher than 25°C. Actinomycin C_1 slightly decreased the retarding activity of CCC. AMC completely neutralized and partially reduced the reversing activity of IAA and coumarin, respectively. It is suggested that in the CCC-affected tissue the rate of synthesis of RNA is slow; IAA and coumarin may reverse the symptoms

of CCC action by inducing and/or acceleration of mRNA(s) synthesis. The primary mode of the CCC-reversing action of IAA is different from that for coumarin.

Phosfon D in a concentration of 10 p.p.m. slightly stimulated growth in the control. In this concentration it markedly reduced both IAA- and coumarin-induced growth. After 8 hours of incubation phosfon D in the concentration of 80 p.p.m. completely blocked growth in the IAA or coumarin treated samples.

B995 at 100 p.p.m. significantly stimulated growth in the control, slightly reduced growth in the IAA affected samples and acted synergistically with coumarin. This compound in the concentration of 200 p.p.m. completely blocked growth of the sections treated with IAA or coumarin; in the latter case very soon visible symptoms of poisoning appeared. It is suggested that B995 can act as the antimetabolite, displacing succinic acid or competing with it in this system.

CCC and phosfon D possibly antagonize the auxin metabolism in this tissue or act at the site of the auxin action. The primary mode of action of each of the growth retardants tested is probably different.

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Działanie CCC, phosfonu D i B995 na stymulowany przez IAA i kumarynę wzrost wycinków hypokotyli słonecznika

Streszczenie

IAA, kumaryna oraz w pewnym stopniu kinetyna odwracają hamujące działanie CCC na wzrost wycinków hypokotyli słonecznika; działanie takiego nie wywiera GA. Efekty wzbudzane przez IAA i kumarynę nie są addytywne; przeciwnie, jeżeli temperatura hodowli przekracza 25°C, to oba stymulatory działają częściowo antagonistycznie. Aktynomycyna C_1, znana jako specyficzny inhibitor syntezy RNA, obniża w pewnym stopniu hamujące działanie CCC oraz całkowicie znosi czynność IAA jako substancji odwracającej zewnętrzne symptomy działania CCC. Można przypuszczać, że w tkankach poddanych działaniu CCC synteza RNA ulega poważnemu zwolnieniu. IAA i kumaryna obniżają hamujące działanie CCC prawdopodobnie dzięki wzbudzeniu lub przyśpieszeniu syntezy RNA. Pierwszorzędny mechanizm działania IAA różni się prawdopopobnie od mechanizmu działania kumaryny przy odwróceniu efektów wzrostowych wzbudzanych przez CCC.

Phosfon D w stężeniu 10 ppm nieznacznie przyśpiesza wzrost wycinków hypokotyli słonecznika, lecz poważnie obniża wzrost wzbudzany przez IAA i kumarynę. Przy stężeniu 80 ppm phosfon D po ośmiu godzinach działania całkowicie hamuje wzrost w seriach zawierających zarówno IAA jak i kumarynę. W tym ostatnim przypadku wycinki ulegają szybkiemu zatruciu.

B995 w stężeniu 100 ppm również przyspiesza wzrost w kontroli; początkowo B995 nie wpływa na wzrost serii poddanych działaniu IAA, następnie notuje się hamowanie; efekty wzrostowe B995 i kumaryny są addytywne. B995 w stężeniu dwukrotnie wyższym (200 ppm) po ośmiu godzinach działania całkowicie blokuje wzrost w seriach zawierających IAA lub kumarynę. Przypuszcza się, że substancja ta działa jako analog strukturalny kwasu bursztynowego.

Stwierdzono więc, że CCC nie działa jak „anty-giberelina" w tym układzie. CCC i phosfon D prawdopodobnie albo zaburzają syntezę auksyn w badanej tkance, albo konkurują z auksynami o to samo miejsce działania (centrum aktywne). Pierwszorzędny mechanizm działania każdego z badanych retardantów wzrostu jest prawdopodobnie różny.

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