RESIDUAL EFFECTS OF AUXIN, CHELATING AGENTS, AND METABOLIC INHIBITORS IN CELL EXTENSION

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Summary

The growth-promoting action of auxin on wheat coleoptile segments can be separated from the act of cellular expansion by use of the technique of Cleland and Bonner (1956). The effects of a pretreatment with auxin ("residual effects") in hypertonic mannitol are manifested later when the coleoptiles are transferred to water. Auxin action, but not water uptake, requires aerobic conditions.

The residual effects of disodium ethylenediaminetetra-acetic acid (EDTA) resemble those due to 3-indolyalactic acid (IAA) in the following respects:

1. Necessity for aerobic conditions during the pretreatment phase.
2. Disappearance of residual effects when the coleoptiles are held in hypertonic mannitol for 150 min.
3. Disappearance of residual effects in presence of an antiauxin (2,4,6-trichloroanisolesalicylic acid).

These facts are held to be contrary to what one might expect if the growth-promoting action of EDTA were attributable to chelation of calcium from the cell wall or other non-metabolic modes of action.

The residual effects due to EDTA, but not those due to IAA, are suppressed by 2,4-dinitrophenol (DNP) at concentrations of 2 mg/l or less. This is taken to mean that the mode of action of EDTA is different from that of IAA and involves certain pathways of oxidative metabolism which may be particularly sensitive to DNP.

As far as they have been investigated the residual effects due to S-carboxymethyl-dimethylthiocarbamate (DTC) resemble those due to IAA both quantitatively and in their interaction with DNP.

In short-term experiments, DNP (a metabolic inhibitor) in very low concentrations may act synergistically with IAA, increasing the growth response to it; like iodoacetate, DNP can also induce auxin-like responses when acting alone. The concentrations which are effective are considerably below those normally used in experiments on the metabolic effects of these inhibitors. In long-term experiments, DNP has an auxin-like action only in the absence of added IAA.

It is suggested that auxin action, whether due to IAA, DTC, chelating agents, or metabolic inhibitors, is due to an induced shift in the metabolic balance of the cell in favour of syntheses and hence of growth. A suitable concentration of any of these substances may promote growth by affecting one of a number of different reactions. Important in this respect are certain steps in metabolism ("pacemaker reactions") at which "branching" occurs and substrates or phosphate esters may be competed for by different metabolic reactions.

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I. INTRODUCTION

Cleland and Bonner (1956) have shown that the process of cell elongation in *Avena* coleoptiles can be separated into two main part processes. The first is dependent on the presence of oxygen and is promoted by auxin. Its main result is believed to be the plasticization of the cell wall. The second process may take place under anaerobic conditions and consists essentially of the uptake of water resulting in cellular expansion. This process is suppressed by hypertonic solutions. The "auxin action" phase can thus be experimentally separated from the "cell expansion" phase. First, the auxin is allowed to act for some time on the tissue which is aerated but prevented from expanding by a suitably high external osmotic concentration. Then the tissue is allowed to expand in water under conditions in which the action of auxin is blocked by a suitable inhibitor, e.g. lack of oxygen. The response of the tissue to auxin in the aerobic phase is expressed as cell expansion in the final anaerobic period. Cell expansion, i.e. water uptake, is very rapid and is not affected by the application of auxin. It is possible with this technique to test, in the final period, the "residual effects" brought about by auxin, effects which are independent of the act of cellular expansion.

The principles underlying this technique were first applied by Heyn (1931) and by Thimann (1951). The method as developed by Cleland and Bonner has been used by Cooil and Bonner (1957) in studying the interaction of calcium and potassium on cell wall plasticity. We have used it to compare the modes of action of chelating agents, which have been shown to have auxin-like activity (see Carr and Ng 1959), with that of auxin.

II. MATERIALS AND METHODS

Segments 10 mm long of coleoptiles of wheat (cv. Gabo) were prepared in the manner previously described (Carr and Ng 1959). Lots of 15 or 20 segments were used for each test solution. Each lot of segments was subjected to three successive periods of treatment. During the first, or pretreatment, period, the segments were immersed for 45 min in 20 ml of 0.3M mannitol buffered with 0.015M K$_2$HPO$_4$–citric acid buffer containing a known concentration of IAA, EDTA, or DTC.* A control without IAA or chelating agent was also set up. In the second, or transition, period the segments were transferred for 30 min to a solution containing 0.3M mannitol (to prevent expansion) and an inhibitor of auxin action. This treatment ensures that the action of auxin is completely blocked before the segments are allowed to expand. During the third, or expansion period, the segments were immersed in 20 ml of buffered glass-distilled water and allowed to expand in the presence of an inhibitor of auxin action for 90 min, after which time there is no further expansion. Then the segments were removed and measured.

Solutions were made up in glass-distilled water. In the experiments the solutions were contained in glass vials and the segments were held in stainless steel wire baskets below the surface of the solutions to prevent the possibility of contact

*The following abbreviations will be used throughout: IAA, β-indolylacetic acid; EDTA, disodium ethylenediaminetetra-acetic acid; DTC, S-carboxymethylidimethylldithiocarbamate; DNP, 2,4-dinitrophenol; 2,4,6-T, 2,4,6-trichlorophenoxyacetic acid.
with air at the surface. During the pretreatment period air was bubbled through the solutions. During the second and third periods anaerobic conditions were maintained (as the inhibitor of auxin action) by bubbling nitrogen through the solutions. In transferring the baskets from one solution to another they were plunged rapidly into the new solution which previously had been saturated with nitrogen. In this manner no more than a few seconds of exposure to air was allowed during the transfers between periods of treatment. Nitrogen was purified by first passing it through two solutions of aged alkaline pyrogallol to absorb oxygen, and then through two scrubbers containing water. Where necessary the gas was passed through a solution of KOH before entering the pyrogallol solution. The bubbling of the test solutions in the transition and expansion periods was commenced at least 40 min before the actual beginning of the transition period. To ensure that the solutions remained saturated with nitrogen a flow of nitrogen was maintained until the end of the experiment.

![Graph](image)

Fig. 1.—Residual effects of EDTA and IAA on extension growth of wheat coleoptile segments. Ordinates: differences ($\Delta L$) between mean lengths of IAA- and EDTA-treated segments after expansion expressed as a percentage of the final lengths of the control segments. Each point is the mean of three experiments, 15-20 segments per treatment. Pretreatment period: aerobic plus mannitol; transition period: anaerobic (nitrogen) plus mannitol; expansion period: anaerobic plus water. All solutions buffered to pH 4.9 with citric acid–phosphate buffer. In Figures 1-10, a vertical line through each point denotes the standard error; points marked S differ significantly at the 5 per cent. level from the controls, which had neither IAA nor EDTA present during the pretreatment period.

The results are expressed as the difference ($\Delta L$) between the average lengths of the treated and non-treated segments after expansion. Significance was estimated at the 5 per cent. probability level, using the $t$-test.

III. Experiments

(a) Residual Effects of IAA and EDTA

The solutions were buffered to pH 4.9. Figure 1 is drawn from the means of three experiments (not those given in Table 1). It is seen that IAA gave a residual effect at 2, 20, and 200 mg/l and EDTA at 2 and 20 mg/l. The differences between the residual effects due to these substances are not significant and although EDTA gave
slightly greater effects than IAA the curves run fairly close together. In one experiment (503, Table 1) both IAA and EDTA gave residual effects at all concentrations. In this experiment, in which the pH was 4·4, the residual effects were much greater than those reported by Cleland and Bonner (1956) for oat coleoptiles and at the highest concentration EDTA produced a very large effect. Initially it was believed that this might have been due to the use on this occasion of an unusually pure sample of nitrogen. On other occasions (e.g. 514, Table 1) "industrial nitrogen" or mixtures of nitrogen and 5 per cent. carbon dioxide (scrubbed as indicated above)

**Table 1**

DIFFERENCE (ΔL) BETWEEN AVERAGE LENGTHS OF TREATED AND UNTREATED (CONTROL) SEGMENTS AFTER EXPANSION

Values in italics differ from the controls (treatment with distilled water, buffered to the stated pH) at the 5 per cent. level

<table>
<thead>
<tr>
<th>Expt.* No.</th>
<th>Agent</th>
<th>pH</th>
<th>Concentration of Agent (mg/l):</th>
<th>ΔL (mm)</th>
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</thead>
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<td></td>
<td></td>
<td></td>
<td>0·002</td>
<td>0·02</td>
</tr>
<tr>
<td>503</td>
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<tr>
<td></td>
<td>EDTA</td>
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<tr>
<td>515</td>
<td>IAA</td>
<td>4·9</td>
<td>0·08</td>
<td>0·03</td>
</tr>
</tbody>
</table>

*Expt. 503, pure nitrogen used. Expt. 514, scrubbed industrial nitrogen used. Expt. 515, industrial nitrogen passed through ammoniacal cuprous chloride and then scrubbed before use.

were used. The possibility was considered that (despite assurances by the manufacturers to the contrary) traces of carbon monoxide might occur in all but the "pure" nitrogen. Experiment 515 (Table 1) was performed to check this. The gas was scrubbed with a carbon monoxide absorber but the responses were not as great as those obtained in the exceptional experiment. It is possible, but not likely, that the differences in pH between experiment 503 and other experiments in this series might account for the difference in responses, but the exceptional result of experiment 503 could not in any way be repeated.

**(b) Residual Effects of IAA and DTC**

At pH 4·4 (Fig. 2) IAA produced significant residual effects at almost all concentrations. With DTC significant residual effects were also obtained at most concentrations and the residual effect of DTC at 20 mg/l was greater than that of
IAA at the same concentration. At pH 7.4 (Fig. 3) and 7.6 residual effects were also obtained, but here a lower concentration was optimal, as was expected from the effects of pH on activity of DTC already described (Ng and Carr 1959). However,

![Residual effects of DTC and IAA on extension growth of wheat coleoptile segments; pH 4.4.](image1)

**Fig. 2.—** Residual effects of DTC and IAA on extension growth of wheat coleoptile segments; pH 4.4. Ordinates: differences ($\Delta L$ mm) between treated and control segments after expansion. Each point represents the mean difference for 15–20 segments.

the large residual effect of DTC at 0.2 mg/l and pH 7.4 is not significantly different from that due to IAA at the same concentration.

![Residual effects of DTC and IAA on extension growth of wheat coleoptile segments; pH 7.4.](image2)

**Fig. 3.—** Residual effects of DTC and IAA on extension growth of wheat coleoptile segments; pH 7.4. Ordinates: differences ($\Delta L$ mm) between treated and control segments after expansion. Each point represents the mean difference for 15–20 segments.

(c) *Aerobic Conditions during the Pretreatment Period Necessary for Residual Effects of IAA and EDTA*

The demonstration of residual effects due to EDTA similar to those due to IAA opens up the possibility of experiments to find out whether they are brought
about in the same way. For instance, auxin will produce residual effects only if the pretreatment phase is aerobic. The results of an experiment in which IAA and EDTA were allowed to act on coleoptile segments in the usual way, but with nitrogen instead of air during the pretreatment period, are shown in Figure 4. The pH was 5.1.

Residual effects were obtained with neither IAA nor EDTA. In fact, some of the segments were slightly shorter at the end of the experiment than at the beginning. From the data of Cleland and Bonner (1956) it is evident that oat coleoptile segments also shrink following a similar treatment. Whether the greater shrinkage following EDTA treatment has any biological meaning is doubtful. In any case it seems clear that, like IAA, EDTA must act in the presence of oxygen in order to produce residual effects.

![Figure 4](image)

Fig. 4.—Residual effects due to IAA and EDTA applied during an anaerobic pretreatment period; pH 5.1. The graphs illustrate the necessity for aerobic conditions during the pretreatment period in order to produce residual effects.

(d) Suppression of Residual Effects by Prolongation of the Transition Period

Prolongation of the transition period appears to suppress the residual effect of IAA (Cleland and Bonner, loc. cit.). Results of experiments to investigate the effect of prolonging the transition period from the normal 30 min to 150 min are shown in Table 2. No significant residual effects were obtained, either with IAA or with EDTA, and in very many treatments, particularly with EDTA, the segments shrank.

(e) Inhibition Due to the Presence of an Antiauxin (2,4,6-T) during Transition and Expansion Periods

According to Cleland and Bonner (1956) the antiauxin 2,4,6-trichlorophenoxyacetic acid exerts a "strictly competitive inhibition of auxin-induced growth in Avena coleoptile sections." Residual effects due to IAA could be partially obliterated
in the presence of 2,4,6-T during the transition and expansion periods. We find that 2,4,6-T at $2 \times 10^{-4}$M can completely obliterate the residual effects due to IAA acting on wheat coleoptile segments in solutions buffered to pH 4·9. The antiauxin was added during the transition and expansion phases. In the presence of this substance no significant residual effects were obtained with any concentration of either IAA or EDTA. Thus the residual effects due to EDTA resemble those due to IAA in their reversibility by 2,4,6-T.

**Table 2**

<table>
<thead>
<tr>
<th>Agent</th>
<th>pH</th>
<th>Concentration of Agent (mg/l):</th>
<th>$\Delta L (\text{mm})$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0·002</td>
<td>0·02</td>
</tr>
<tr>
<td>IAA</td>
<td>4·9</td>
<td>-0·05</td>
<td>-0·20</td>
</tr>
<tr>
<td>EDTA</td>
<td>4·9</td>
<td>-0·25</td>
<td>-0·09</td>
</tr>
<tr>
<td>IAA</td>
<td>5·0</td>
<td>0·02</td>
<td>0·01</td>
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<tr>
<td>EDTA</td>
<td>5·0</td>
<td>-0·04</td>
<td>-0·04</td>
</tr>
</tbody>
</table>

(f) Effects Due to DNP during the Transition and Expansion Periods

In seeking differences in the mechanism by which EDTA and IAA bring about the residual effects described above, DNP was used as the inhibitor of auxin action in the transition and expansion phases instead of nitrogen. The concentration used was 2 mg/l, which Cleland and Bonner (1956) have shown not to prevent the appearance of residual effects due to IAA. The results of experiments with IAA and EDTA at pH 4·5 and 5·5, and with IAA and DTC at a pH of 7·6 are summarized in Figures 5 and 6.

The results confirm the statement of Cleland and Bonner that 2 mg/l of DNP does not suppress the residual effects due to IAA, since all but the lowest concentration of IAA gave significant residual effects. On the other hand, EDTA either produced no effects at all or small effects with no proportionality to concentration. The clear and obvious difference in behaviour of the residual effects due to IAA and EDTA in the presence of DNP indicates that the mode of action of EDTA on the growth of the coleoptile must differ in some essential manner from that of auxin. At pH 7·6 IAA again gave large residual effects but the relationship to IAA con-
centration was somewhat erratic, 2 mg/l producing particularly large responses (Fig. 6). DTC gave residual effects at the higher concentrations rising to a maximum at the highest concentration, and the responses were almost as great as those induced by IAA at equivalent concentrations.

(g) Action of very low Concentrations of DNP on Residual Effects of IAA and EDTA

Although DNP at 2 mg/l discriminates against the residual effects of EDTA in cell extension, it might be argued that this would not occur with lower concentrations of DNP. To test this a range of low concentrations of DNP was applied during the transition and expansion periods, while air was bubbled through the solutions. Only one concentration of IAA or of EDTA was used (2 mg/l) and the solutions were buffered to pH 4.5. The results, shown in Figure 7, were surprising in that even with very low concentrations of DNP there were no residual effects of EDTA. On the other hand, IAA produced quite large residual effects in the presence of DNP at 2 mg/l or less but not at 5 mg/l. Indeed, as can be seen from Figure 7, the residual effect of IAA in the presence of 1 mg/l DNP was greater than that induced by IAA alone with no DNP added during the transition or expansion periods. Two conclusions may therefore be drawn from the data. Firstly, support is given to the view that the mechanism of action of EDTA is radically different from that of

![Figure 5](image-url)
IAA. Secondly, a metabolic inhibitor, DNP, in very low concentration appears to act synergistically with IAA, increasing the growth response to it.

(h) Residual Effects of DNP and Iodoacetic Acid

If DNP can enhance the response to IAA then it may be able to stimulate growth in the absence of added auxin, and to produce residual effects. In testing this, different concentrations of DNP were used in the aerobic pretreatment period and nitrogen was bubbled through the solutions (buffered to pH 5·0) during the remaining periods. DNP was present only during the pretreatment period. As may be seen from the results (Fig. 8), DNP either had no effect or was slightly inhibitory at the higher concentrations (0·1 mg/l and above) but at 0·001 and 0·01 mg/l it actually caused a stimulation of growth.

![Graph showing residual effects due to IAA and DTC with the inhibitor DNP (2 mg/l) applied instead of nitrogen during the transition and expansion periods; pH 7·6.](image)

With previous reports (e.g. Slocum and Little 1957) of growth stimulation by low concentrations of another metabolic inhibitor, iodoacetate, in mind we have sought to determine whether iodoacetate would also induce residual effects. The method was the same as that described for DNP, except that the solutions were buffered to pH 4·8. The results (Fig. 9) indicate that, like DNP, iodoacetate can also produce residual effects in the absence of an exogenous supply of auxin. The stimulatory range appears to be small and the optimum around 0·002 mg/l. Neither this nor the low concentrations of DNP which can induce residual effects on growth are generally considered to be effective in the inhibition of metabolic processes.
(i) Effects of Low Concentrations of DNP in the Normal Straight-growth Test with Wheat Coleoptiles

In view of the surprising result described above some low concentrations of DNP were tested in the normal straight-growth test. The technique used was that already described (Ng and Carr 1959) with the exception that the incubation time was 22 instead of 24 hr. The solutions were buffered to pH 5.0. The results are shown in Figure 10. When auxin (0.2 mg/l) was added to the solutions, all concentrations of DNP were inhibitory. In the absence of added auxin, however, the lowest concentrations of DNP, with an optimum at 0.01 mg/l were found to promote growth.

![Graph showing the effects of DNP on growth](image)

Fig. 7.—Residual effects due to IAA and EDTA (both at 2 mg/l) at pH 4.5 with DNP at different concentrations applied during the transition and expansion periods. Upper graph, IAA applied during the pretreatment period, aerobic conditions during transition and expansion periods. Significance (8) is with respect to the control (lower graph) with neither IAA nor EDTA in pretreatment period. Note (1) synergism of DNP (at 1 mg/l) with IAA at 2 mg/l; (2) complete blockage of residual effects due to EDTA at all concentrations of DNP.

IV. DISCUSSION

The data corroborate the findings of Cleland and Bonner (1956) concerning the residual effects due to IAA but also shed light on the nature of growth-promotion by chelating agents such as EDTA. Although the residual effects due to EDTA are very similar in many respects to those induced by IAA (requirement for aerobic conditions in the pretreatment period, obliteration by 2,4,6-T, or by prolonged immersion of the segments in hypertonic mannitol) it does not follow that the modes
of action of these substances are identical. In fact, the mode of action of EDTA is differentiated from that of IAA by its response to the presence of DNP. In previous papers (Carr and Ng 1959; Ng and Carr 1959) we have examined the effects of chelating agents on coleoptile growth in the light of the hypothesis (Heath and Clark 1956a, 1956b) that these substances sequester calcium from the cell wall, thus increasing its plasticity. It is abundantly clear from the data given above that this hypothesis cannot explain the mode of action of EDTA on growth. It cannot explain, for instance, the dependence of the residual effects due to EDTA on oxidative metabolism. The possibility that calcium may be involved in the “stiffening” of the cell wall during a prolonged transition period has been raised by the work of Cooil and Bonner (1957) but on any hypothesis that EDTA chelates calcium from the cell wall the residual effect of EDTA should persist over a prolonged transition period.

Fig. 8.—Residual effects due to pretreatment with DNP; pH 5.
Nitrogen applied during transition and expansion periods.

On the other hand, it is difficult to explain why the antiauxin, 2,4,6-T should be able to reverse the plasticization of the cell wall caused either by IAA or by EDTA, even under anaerobic conditions (in which neither IAA nor EDTA is effective). No explanation of the mode of action of this antiauxin has been put forward by Cleland and Bonner (1956). It is difficult to imagine that it merely stimulates demethylation of pectins or the formation of calcium pectate because it is effective even when present only during the expansion phase. The fact that suitable concentrations of DNP in the transition and expansion periods can obliterate “residual” effects which have already been completed or at least initiated in the pretreatment period may mean that the persistence of the residual effects during the transition period must depend on persistence of the train of metabolic events initiated by the growth-promoting substance.

Unlike that of IAA, the action of EDTA appears to depend on phases of metabolism which are very sensitive to DNP. It should be stressed, however, that this conclusion applies only to the short-term experiments. In longer experiments, lasting 22 hr or more, DNP at 2 mg/l, a concentration which is ineffective in short-term experiments, is inhibitory to growth. At least two explanations of this are
possible. In Stenlid's (1949) experiments the carrot leaves treated with $10^{-4}$ M DNP at pH 5 took up more oxygen than the controls over the first hour, but after that time oxygen uptake was strongly inhibited. On the other hand, Thimann and Bonner (1948) have shown that coleoptiles become, with age, more sensitive to inhibition by iodoacetate. Some such aging phenomenon may be responsible for inhibition, in experiments of long duration, by DNP concentrations which are not inhibitory in the residual-effect experiments. Thimann and Bonner also confirmed the statement of Commoner and Thimann (1942) that, in the presence of sucrose and IAA, iodoacetate promotes the growth of oat coleoptiles. According to Slocum and Little (1957) iodoacetate and arsenite stimulate growth by partially inhibiting the

![Graph](image)

Fig. 9.—Residual effects due to iodoacetic acid; pH 4.8. Nitrogen applied during transition and expansion periods.

utilization of pyruvate or α-ketoglutarate in energy production, thus releasing more hexose carbon to the synthesis of structurally essential compounds. This redistribution is dependent on the auxin concentration because "at the optimal concentration of auxin we have an ideal dynamic balance at which the division of substrate carbon between the two processes of energy production and utilization for structure is precisely optimal". Some such scheme as that proposed by Slocum and Little may explain the stimulatory effects of DNP on growth, if, in low concentration, this compound enables a new dynamic equilibrium in the utilization of hexose to be set up in the growing cell. It may be supposed that with 20 mg/l of IAA in the medium and other conditions "normal" the metabolic balance of wheat coleoptile segments is optimal for growth. If DNP is added at, say $10^{-7}$ M (a concentration which has no, or only stimulatory, effects on oxygen uptake according to Simon (1953) and French and Beevers (1953)) the greater utilization of hexose in energy production may inhibit growth (as shown in Fig. 10) but stimulate respiration. In
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the absence of added auxin, however, the same concentration of DNP might shift the balance of metabolism in favour of growth. The more or less complete uncoupling of oxidative phosphorylation resulting from comparatively large concentrations of DNP would be quite unfavourable for growth, since synthetic reactions could not then proceed. The level of auxin necessary so that stimulation by DNP may be displayed must be rather low, and is probably about the normal endogenous level in coleoptile segments. This may well be the explanation of the difference in response of different

kinds of tissues of *Parthenocissus* and sunflower to "concentrations of DNP which are not known to affect respiratory processes or the accumulation of ATP" reported by Klein (1957). Habituated and callus tissues showed stimulation of growth by concentrations between $10^{-10}$ and $10^{-7}$M, but crown gall tissues, which are known to be rich in growth hormones, showed only inhibition by DNP at all concentrations between $10^{-11}$ and $10^{-6}$M. Klein has offered no explanation of his results, but they appear to agree remarkably well with those described above for coleoptile segments.

Opinions differ on the ability of DTC to act as a complexing or chelating agent. Wain (private communication) believes that it has little or no such activity. The general opinion, however, is that it elicits true auxin responses (see Ng and Carr 1959) and this is supported by the data presented in this paper. The fact that DTC gives large residual effects which, like those due to IAA, are insensitive to DNP at 2 mg/l,
strengthens the impression that its mode of action is closely similar to that of IAA. It is possible that DTC and IAA may act on one or more of the “pacemaker” reactions (Krebs 1957), the stages of cell metabolism most “liable to be influenced by inhibitors or hormones” thereby bringing about a subtly balanced utilization of carbohydrate which is favourable to growth processes. Chelating agents may also affect pacemaker reactions and thus, indirectly, act as growth-promoting substances. Probably they intervene at the electron-transfer stage where they have been shown to have effects on respiration (James 1953).

Since both respiratory and synthetic processes in the cell are often dependent on the level of phosphate esters it is possible that the effects of growth-promoting substances are due partly to effects on the level of phosphate esters. For instance, although the role of ATPase in the cell is still uncertain, DNP, EDTA, and Ca++ are all known to have marked effects on this enzyme in mitochondrial preparations (Forti 1957). Stimulation and inhibition, by different concentrations of thyroxine, of syntheses dependent on ATP in animal cells have been explained by Hoch and Lipmann (1953) in terms of changes in the rate of turnover of phosphate acceptors. Too tight or too loose a linkage between respiration and phosphorylation is “undesirable” and optimal concentrations of thyroxine maintain a “favourable” balance. In view of the rather common phenomenon of an “optimum concentration” which many substances display (e.g. IAA, DNP) when they both stimulate and inhibit a specific process, it may be suggested that they act in metabolism in a way similar to that envisaged by Slocum and Little (1957) whose scheme is a specific realization of the proposals of Hoch and Lipmann and of Thimann (1956).

As Audus (1954) has remarked, the trend in auxin physiology has been to seek a “master reaction” controlled by auxin. There may in fact be no single “master reaction”. A suitable concentration of any metabolite or poison or other effective agent whether auxin, chelating agent, metabolic inhibitor, or substrate, which is able to shift the balance of metabolism of the cell (which must at the outset be potentially capable of growing), in favour of synthesis of protein or of wall components will be growth-promoting. The number of ways in which this might be achieved could be large, although certain critical “pacemaker” reactions may be particularly susceptible.

V. ACKNOWLEDGMENTS

We are indebted to Mr. J. M. Robinson, I.C.I.A.N.Z., Merrindale Research Station, Victoria, for a gift of 2,4,6-T.

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VI. REFERENCES


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