and that they were active to a proportionately greater extent in the low nitrogen treatment. The results show that in experiments using tap water, and possibly also where distilled water is used, the level of nitrogen present in the plant does not necessarily bear a relation to the amount supplied to the plants in the nutrient solution, and may explain why few workers have been able to detect an effect of nitrogen nutrition on flower initiation.

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Promotion of Cell Wall Synthesis by Indolylacetic Acid

IT is widely believed that the increase in cell wall synthesis which occurs when growth of plant cells is promoted by auxin is neither a direct consequence of hormone action nor a causal factor in cell enlargement, but is induced indirectly by the occurrence of cell enlargement[1]. A principal reason for this view is that if elongation is inhibited osmotically by mannitol, wall synthesis becomes reduced[2], and promotion of wall synthesis by auxin does not occur[3].

Table 1 compares the effect of 3 mg/l. indolylacetic acid (IAA) on cell wall synthesis in oat coleoptile segments: (*a*) when no inhibitor is present and elongation occurs; (*b*) when elongation is inhibited by 0·25 M mannitol; (*c*) when elongation is inhibited by 0·01 M calcium chloride, which is considered to prevent elongation by making the cell wall more rigid[4]. Segments 8 mm. long were cut from coleoptiles of 3-day-old oat seedlings, var. Victory. Groups of eight segments were floated in 1·0 ml. of 0·05 M glucose containing 1·67 μc. of glucose uniformly labelled with carbon-14, on a reciprocating shaker at 120 oscillations per min. in the dark. After 7 h the segments were washed with water, measured and placed in 1 ml. of 80 per cent ethanol to extract soluble radioactivity. Segments were then crushed between glass plates (as described in ref. 5), and extracted with 0·2 per cent crystalline pepsin in 0·03 M phosphate buffer, *p*H 2, for 24 h to remove protein. The insoluble residues, representing cell wall material, were dried on planchets and counted with 25 per cent efficiency. Absorbed activity, as given in Table 1, is the sum of extractable activity and activity incorporated into the cell wall material.

When elongation is not inhibited, IAA causes a large increase in wall synthesis, as has been found previously (ref. 5, and literature cited there). This is associated with an increase in uptake of glucose, but the percentage of the absorbed glucose which is incorporated into the cell wall is nevertheless increased substantially by IAA.

In the presence of 0·25 M mannitol, no promotion of cell wall incorporation by IAA can be demonstrated, in agreement with earlier findings[3].

However, when elongation is inhibited instead by calcium ions, a conspicuous promotion of cell wall incorporation by IAA is observed, associated with no increase in uptake of activity. A similar IAA promotion of incorporation in the presence of calcium ions can be demonstrated in *p*H 4·5 maleate buffer, so the effect does not appear to be due to differences in *p*H.

Table 1. EFFECT OF IAA ON CELL WALL SYNTHESIS

Inhibitor	IAA	Length (mm.)	Cell wall activity (c.p.m.)*	Absorbed activity (c.p.m.)*	Percentage incorporated †	IAA effect‡
None	–	9·2	8,470 ± 820	55,420 ± 3,970	15·3 ± 0·9	
	+	11·8	14,540 ± 410	68,450 ± 4,290	21·2 ± 0·9	1·38
Mannitol	–	8·8	6,000 ± 780	48,540 ± 5,440	12·4 ± 1·2	
	+	8·7	5,460 ± 200	42,830 ± 2,850	12·7 ± 0·8	1·03
$CaCl_2$	–	8·7	7,540 ± 90	43,760 ± 1,480	17·3 ± 0·8	
	+	8·8	10,760 ± 130	43,120 ± 3,210	25·0 ± 1·6	1·44

* Mean and average deviation for 4 replicate samples each consisting of four coleoptile segments.
† Percentage of absorbed activity which was incorporated into cell wall.
‡ Percentage of incorporation in presence of IAA, divided by percentage incorporation in absence of IAA.

The experiment with calcium ions shows that auxin has a promotive effect on cell wall synthesis which is direct in the sense that it is not a result of the effect of auxin on elongation. That IAA promotion cannot be detected when elongation is inhibited by mannitol indicates that mannitol inhibits this action of IAA on wall synthesis.

Relative to uptake of glucose, the promotion of wall incorporation by IAA in the presence of calcium ions is similar to the IAA promotion which is observed in the absence of inhibitor, when rapid elongation is occurring (Table 1, last col.). We presume, therefore, that the same direct IAA promotion is operating during elongation, but is being added to by an indirect effect which may result from elongation and is associated with an increase in uptake of glucose.

The promotion of total wall synthesis by IAA in the presence of calcium ions is, by per cent, comparable with the promotion of hot water soluble pectin synthesis by IAA, which has been emphasized as involved in the action of auxin on elongation[6]. While hot water soluble polyuronides constitute less than 1 per cent of the cell wall, the presently described promotion involves cell wall synthesis as a whole and is, therefore, a gross effect compared with that on pectin. It is due mainly to increases in incorporation of sugar constituents of the cell wall material rather than uronic acids. Detailed information on the effect of IAA on incorporation into individual constituents will be published elsewhere.

The magnitude of the effect of IAA on cell wall synthesis, demonstrable in the presence of calcium ions, suggests that this effect could be causally related to the effect of IAA on elongation. This possibility will be explored.

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