As with other gibberellin reactions, the minimum active concentration for the induction of antheridia in *Anemia* depends greatly on the type of gibberellin applied. So gibberellin *A*7 is still active in a concentration of 5×10^{-10} g/ml. which is 10^{-5} of the minimum active concentration of gibberellin *A*5 or *A*8.

The order of activity for the induction of antheridia in *Anemia phyllitidis* was found to be:

$$A7 > A4 \geqq A1 > A3 \geqq A9 > A5 \geqq A8$$

This sequence is different from those described for gibberellin reactions known so far. Details of these results, including results on the effectiveness of antheridium formation in polipodiaceous ferns, will soon be published.

The gibberellin *A*3 used was obtained from Böhringer and Söhne, Mannheim, gibberellin *A*1 from Abbott Laboratories, North Chicago. Samples of gibberellin *A*4, *A*5, *A*7, *A*8 and *A*9 were given by Imperial Chemical Industries, Ltd., Welwyn, through the good offices of Dr. P. W. Brian and Dr. J. Macmillan.

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Accelerated Release of Ethylene by Cotton following Application of Indolyl-3-acetic Acid

PREVIOUSLY we reported that 2,4-dichlorophenoxyacetic acid (2,4-D) stimulated the release of ethylene by the cotton plant (*Gossypium hirsutum*)[1]. The maximum stimulation occurred during the initial period of growth responses to 2,4-D and before chlorosis or necrosis was visible. This report presents evidence that the response was not peculiar to the highly phytotoxic 2,4-D, but that a native auxin, indolyl-3-acetic acid (IAA), also accelerates the release of ethylene.

Deltapine-*TPSA* cotton plants, 35 days old, were divided into two groups and one lot was sprayed with $1{\cdot}4 \times 10^{-2}$ m.moles IAA per plant. Since previous experiments revealed that the carrier solvent (aqueous 10 per cent ethanol with 1 per cent 'Tween 20') would not significantly influence ethylene production by cotton, the control plants were not sprayed. Lighting, plant chambers, air flow, ethylene collection and measurement followed the earlier procedures[1]. The temperature in the plant chambers was maintained at 22 ± 1° C at night and 24 ± 1° C during the light period.

The data show that IAA does stimulate the release of ethylene by cotton plants (Table 1). This conclusion was verified by a subsequent experiment with slightly older plants. The first experiment was continued for three additional 48-h periods during which the treated plants produced small, but detectable, amounts of ethylene. The amount of IAA applied was sufficient to cause epinasty, twisting and declination of the apical stem and puckering of most of the leaves. The severity of symptom expression declined during the second collection period after treatment. At the end of this period most of the plants had straightened up to their original height.

Table 1. EFFECT OF IAA ON THE RELEASE OF ETHYLENE BY COTTON

Treatment	μl. Ethylene/24 h/kg fresh weight: First collection period (48 h)	Second collection period (54 h)
IAA	128	26
Control*	0	0

* Any ethylene produced was below the limits of sensitivity of the method.

During the second period the release of ethylene declined sharply, apparently being correlated with the visible recovery. There was no visible chlorosis, necrosis or other toxic effects from IAA during the experiments.

The present experiments will not allow a critical comparison of the relative activity of IAA and 2,4-D in stimulating ethylene synthesis by cotton. The application of IAA was 5·4 times greater than the previous applications of 2,4-D[1]. On this basis, IAA appears less effective for a shorter period of time than 2,4-D. This apparent lower activity of IAA could indicate *in vivo* activity of IAA oxidase in cotton[2] as compared with the relatively slow breakdown and complexing of 2,4-D in the same species[3].

It is significant that an exogenous application of a naturally occurring auxin stimulates the synthesis of a physiologically active gas (Table 1) which will also induce auxin-like symptoms[4,5]. This suggests that some responses previously attributed solely to auxin may actually be due to abnormal tissue levels of ethylene and/or that both regulators have parallel modes of action in several plant systems.

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Nucleotide Synthesis in Hazel Seeds during After-ripening

SEED dormancy of the type shown by sour cherry (*Prunus cerasus* L.) and hazel (*Corylus avellana* L.) appears to be a widespread phenomenon. Before such seeds can germinate at room temperature they have to undergo a process of after-ripening at a low temperature (for example, 5° C) in the presence of an adequate supply of moisture. Olney and Pollock have demonstrated that during after-ripening in sour cherry seeds there is an increase in the concentration of phosphorus, mainly in the nucleotide and nucleic acid-containing fractions, in the cells of the potentially growing organs of the seed[1]. They have suggested that cherry seed dormancy may be associated with a block in the phosphate metabolism of the cells, although they qualify their conclusion by stressing the difficulty of distinguishing the primary reaction responsible for the breaking of dormancy from the many secondary reactions.

In experiments with hazel seed, we have obtained evidence which supports the conclusion of Olney and Pollock and which gives further insight into the mechanism of after-ripening. Adenine-8-^{14}C was supplied to cotyledon slices and embryonic axes taken from hazel seeds after various periods of after-ripening at 5° C. In each case the labelled adenine was supplied at 5° C for 24 h, after which the distribution of label was determined. Adenine, adenosine, adenosine-5′-monophosphate (AMP) and adenosine-5′-diphosphate (ADP) accounted for most