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Ethylene, Plant Senescence and Abscission[1]

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Abstract. **Evidence supporting the hypothesis that ethylene is involved in the control of senescence and abscission is reviewed. The data indicate that ethylene causes abscission *in vivo* by inhibiting auxin synthesis and transport or enhancing auxin destruction, thus lowering the diffusible auxin level. Studies with isolated leaves and explants suggest that the gas also may influence abscission by accelerating senescence and through an action on plant cell walls. Freshly prepared explants produce ethylene at a rate which must be high enough to maximally affect the tissue and this may explain why these explants (stage I) cannot respond to applied ethylene.**

In the period between 1860 and 1870 it was reported on several occasions that certain trees (34, 49) and many varieties of plants (30) were defoliated after accidental exposure to illuminating gas. That ethylene contained in illuminating gas caused the leaf fall was suggested by the demonstration in 1901 (58) that the olefin is the biologically active component of illuminating gas, and directly proven by Doubt in 1917 (29). During the next 4 decades the ability of ethylene to induce abscission was considered, as were most other actions of the gas, to be merely an interesting and remarkable curiosity. However, it now has been established that the gas is an endogenous regulator not only of fruit ripening (12, 14), but also of vegetative (17, 21, 24, 35, 36, 48) and reproductive activities (16, 23). Therefore, it is appropriate to inquire whether ethylene also controls or influences the natural abscission process.

Behavior of Whole Plants Exposed to Ethylene. Applied ethylene is most effective in causing old leaves to abscise so that as plants age, progressively less of the gas is required to defoliate them (29). Similarly older leaves are more prone to abscise than young ones when defoliants are applied (51). This susceptibility of old leaves is correlated with and in fact may be due to their low auxin content (69), and thus it is not surprising that application of NAA or IAA prevents ethylene from stimulating abscission both in intact plants (28, 39) and explants (2, 5). We have noted that applied auxin (2, 4-D) also prevents ethylene from inhibiting growth in light grown pea plants, and deduce from this that ethylene might depress growth by reducing this plant's auxin content. Possibly a similar action causes ethylene induced abscission *in vivo*.

Effect of Ethylene on Auxin Metabolism. When ethylene is applied to intact plants, the amount of diffusible auxin which can be recovered is considerably reduced (37, 50, 55, 68). There are several possible explanations for this response: (i) *Ethylene inhibits polar transport*: Although ethylene has no effect on auxin uptake or polar auxin transport when it is applied to excised stem sections (1, 19, 50, 55), it often inhibits transport *in vivo* (19, 37, 56, 57). The capacity of the transport system in the stem of an etiolated pea plant is reduced by over 90 % within 24 hours after ethylene is applied, without any significant change in the velocity of the system (19). We could not detect a transport disturbance in coleoptiles cut from *Avena sativa* seedlings which had been exposed to and responded to ethylene during a 24 hour period; but the transport capacity of corn coleoptiles was reduced 30 % under the same conditions, indicating that the gas is able to disturb transport in this modified leaf (19). In all cases the inhibition is irreversible, persisting long after ethylene is removed. (ii) *Ethylene enhances auxin destruction*: Morgan *et al.* (39, 56) found that ethylene fumigation enhances IAA oxidase activity in cotton and several other plants. Although this cannot account for the lowered diffusible auxin level of pea and *Avena* seedlings exposed to ethylene, because auxin destruction is not significantly altered in these cases (17, 19, 56), it could be an important factor in other tissues. It has been suggested that ethylene induced IAA oxidase activity might mediate ethylene stimulated abscission in cotton (66), and this idea is supported by the fact that phenols which stimulate IAA oxidase activity promote abscission of cotton explants, whereas those which inhibit IAA oxidase activity retard abscission. (iii) *Ethylene inhibits auxin*

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synthesis: Valdovinos *et al.* (68) report that the conversion of labeled tryptophan to IAA by breis derived from light grown pea and *Coleus* plants is much reduced if the plants have been treated with 25 ppm ethylene for 18 hours.

Any or all of the above responses could account for the loss of diffusible auxin in ethylene treated tissue and thus explain why the gas stimulates abscission.

Epinasty vs. Abscission. Epinasty, unlike abscission, is not prevented by auxins; to the contrary high concentrations of auxin *induce* epinasty by stimulating ethylene formation (39). Therefore, even if the endogenous auxin level were sustained at a high enough value to prevent abscission, leaves still ought to become epinastic in the presence of ethylene. Doubt (29) and others (27, 40) report that exposure to 0.1 to 2 ppm ethylene produces epinasty without causing any leaves to fall, whereas the same treatment carried out with 2 to 10 ppm ethylene defoliates all but the youngest leaves. These results suggest that a low concentration of ethylene may induce epinasty without substantially reducing the auxin level, while a higher concentration could be required to lower the auxin level sufficiently to permit abscission. Such an interpretation would explain why abscission *in vivo* seems to require more ethylene than is needed to produce the same response in explants, or other responses both *in vivo* and *in vitro* (table I).

Production of Ethylene in Vivo. Ethylene is usually produced in those parts of the plant which have the highest auxin content, presumably because the production of the gas is stimulated by auxin (2, 6, 16, 17, 21, 23, 24, 39, 42, 48, 62). A high rate of ethylene evolution is associated with the scale leaves and apical regions of the stem and root of etiolated pea seedlings (21, 35, 48), also with the apex and youngest petiole of a light grown pea stem (fig 1); in contrast an older petiole produces much less of the gas. Thus throughout the etiolated pea plant the rate of ethylene production is correlated

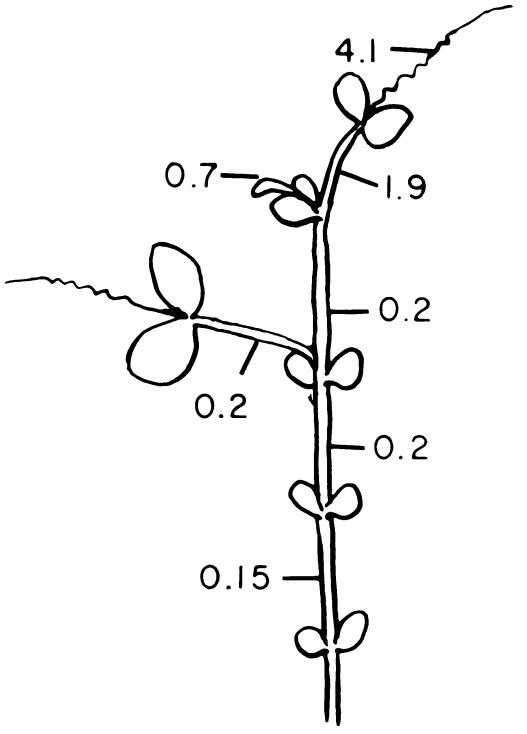

FIG. 1. Ethylene production by various parts of 14 day old green pea plants. Rates were determined for 5 mm pieces of stem and petiolar tissue, or entire tendrils and apical hooks. Tissue was incubated in a solution containing 2% sucrose and 50 mM potassium phosphate buffer (pH 6.8) until the wound response had subsided (5 hr), and then 20 sections were sealed in 50 ml Erlenmeyer flasks containing 8 ml of the same solution to determine ethylene evolution during an additional 18 hours.

Table I. *Relative Sensitivities of Various Processes to Ethylene*

Response	Ethylene conc Threshold	Half-maximal	Maximal
	ppm	*ppm*	*ppm*
Inhibition of hook opening	0.01	0.1	1
Stem swelling and inhibition of stem growth	0.01	0.2	2
Root swelling and inhibition of root growth	0.01	0.25	3
Epinasty	0.025–0.05	...	1
Inhibition of lateral IAA transport	0.03	0.3	1
Leaf fading	0.02	0.3	2
Abscission of explants	0.01	0.15	1
Inhibition of bud growth	...	0.2	2
Fruit ripening	0.1–0.2	...	1–10

[1] These data are taken from references 2, 12, 17, 21, 24, 27, 35, 48.

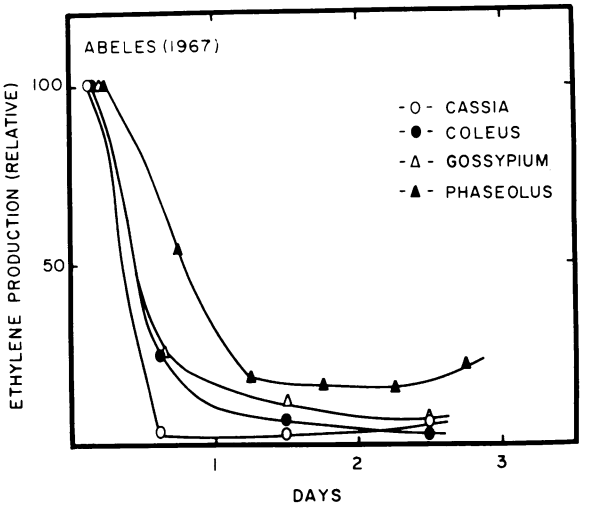

FIG. 2. Progressive changes in the amount of ethylene produced by freshly prepared explants (adapted from ref. 2). All rates are compared to that measured during an initial 6 hour period, which is considered to be 100 % in each case.

with a high auxin content (59) and low IAA oxidase activity (33). When the IAA content is artificially altered by applying growth hormone, the rate of ethylene production continually reflects the level of free IAA. For example, ethylene production by pea roots (24) increases abruptly within 15 to 30 minutes after IAA is applied, reaches a maximum coincident with free IAA within a few hours, declines, and stops in about 10 hours when all IAA has been

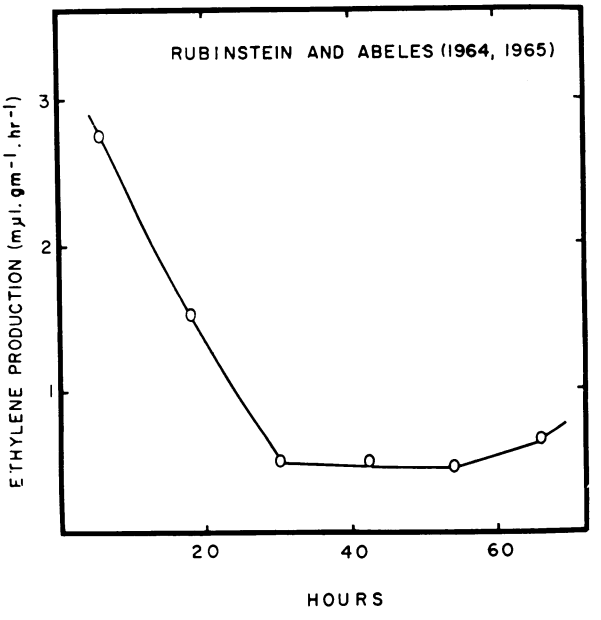

FIG. 3. Progressive changes in the absolute rate at which ethylene is produced by freshly prepared bean explants (adapted from ref. 62, assuming the tissue to contain 80 % water on a fr wt basis).

destroyed or converted to indoleacetyl aspartate (8). The magnitude of the stimulation which auxin causes also depends upon the age of the tissue. Thus the capacity of bean petiole explants to produce ethylene when NAA is applied decreases with time (6), and we find that while the threshold concentration of IAA needed to elicit ethylene production in various parts of the etiolated pea stem is fairly constant, the magnitude of the response at each IAA concentration is inversely related to the age of the tissue.

Behavior of Explants Exposed to Ethylene. Explants derived from *Phaseolus* and other plants produce ethylene at a maximal rate immediately after they are excised, and then the rate declines (2 and fig 2). Ethylene production per gram fresh weight of bean explants can be calculated from published data (62), and is presented in figure 3. These values only become meaningful when we inquire "how low a rate of ethylene production suffices to cause abscission"?

Relationship Between Ethylene Production Rate, Applied Ethylene, and the Internal Ethylene Content. Gas exchange in plant and animal tissue is regulated by Fick's law. At equilibrium the rate of ethylene production equals the rate at which the gas escapes from the tissue, and this in turn is proportional to a number of constants (surface area, volume, diffusivity of ethylene, *etc.*) times the concentration gradient forcing ethylene from the tissue into the ambient air (15). Thus:

rate of production = rate of escape = $K\ (C_{in} - C_{out})$

where K is a constant where C_{in} the concentration of ethylene within the tissue, C_{out} the concentration in the ambient air, and $C_{in} - C_{out}$ is proportional to the concentration gradient. The action of ethylene depends only on the internal concentration (C_{in}), and if a tissue is well aerated ($C_{out} = 0$) the internal concentration is directly proportional to the rate of ethylene production. When a tissue is not well aerated or if ethylene is applied, the internal content is increased by exactly the amount of gas applied or accumulated; so that ($C_{in} - C_{out}$) always equals the internal concentration of well aerated tissue.

It is unfortunate that many studies on ethylene production simply measure the concentration of ethylene in the gas phase above tissues sealed in closed containers. Obviously if sufficient ethylene accumulates it will be the dominant factor determining the internal concentration, but this seldom is the case. For example, by direct measurement it can be shown that a gram of apple tissue has to be sealed in a 100 ml bottle for 200 to 1000 hours in order to double its internal concentration relative to that of well aerated tissue (12, 15). By indirect methods (see below) it can be calculated that the internal concentration of ethylene in a gram of vegetative tissue would double under the same conditions during about 40 hours confinement. It makes no difference what the absolute rate of ethylene evolution is in such a case; all that matters is the

relative amount of tissue and size of the bottle. Therefore measurements of ppm ethylene in the gas phase vastly underestimate the internal ethylene concentration and are not directly proportional to it under normal experimental conditions.

A quantitative relationship between internal ethylene content and the rate of ethylene production has been derived by direct measurement in the case of fruit tissues (12, 15), and indirectly with vegetative tissue (14, 16, 17, 24). A few ppm and all higher concentrations of ethylene applied to etiolated pea stem sections cause maximal swelling and inhibition of elongation, and if the external IAA concentration is raised to a level that induces ethylene production at not less than 5 $m\mu l \cdot gm^{-1} \cdot hr^{-1}$, maximal swelling and inhibition of elongation result from endogenously produced gas (17). Under these conditions the tissue becomes unresponsive to applied gas. In other words pea tissue behaves as though it contains a few ppm ethylene when it produces the gas at a rate of 5 $m\mu l \cdot gm^{-1} \cdot hr^{-1}$. Identical values have been obtained with sunflower stem tissue (17), and pea roots (24). Similarly the hook of an etiolated black Valentine bean plant is prevented from opening in red light by 1 ppm applied ethylene (table I), and normally remains almost completely closed when it produces ethylene at a rate of 3.4 $m\mu l \cdot gm^{-1} \cdot hr^{-1}$ (48). In each case a rate of ethylene production in the range between 3 and 5 $m\mu l \cdot gm^{-1} \cdot hr^{-1}$ causes the tissue to respond completely to its endogenous gas, and since abscission in explants is maximally stimulated by the same amount of ethylene required to affect hook closure and the swelling response (table I), it follows that a rate of ethylene production between 3 and 5 $m\mu l \cdot gm^{-1} \cdot hr^{-1}$ should be completely effective in accelerating abscission. As freshly prepared bean explants produce 3 $m\mu l$ of ethylene per $gm^{-1} \cdot hr^{-1}$ (fig 3) they must be optimally stimulated.

Significance of Endogenous Ethylene Production in Explants. Rubinstein and Leopold (63) defined stage I explants as those whose longevity is extended by application of auxins. When the explant ages in the absence of applied auxin for 6 to 12 hours it enters a new phase, stage II, during which applied auxins, amino acids and ethylene stimulate abscission (fig 4; refs 2, 26). The idea that a certain aging process must occur before petioles become sensitive to ethylene implies that the gas does not accelerate stage I, and this hypothesis is supported by data showing that applied ethylene fails to cause chlorophyll breakdown, protein hydrolysis, and loss in dry weight of bean pulvinar tissue (5). Osborne (43) holds an opposing view, that ethylene enhances senescence in the petiole just as it does in certain other tissues (23, 29, 53, 70), and results obtained by Dijkman and Burg (fig 5 and table II) support this interpretation. Ethylene enhances degreening of isolated *Avena* leaves; about 0.3 ppm is half effective, and a 3 to 6 hour exposure to 2 ppm and all higher concentrations produces a maximal effect.

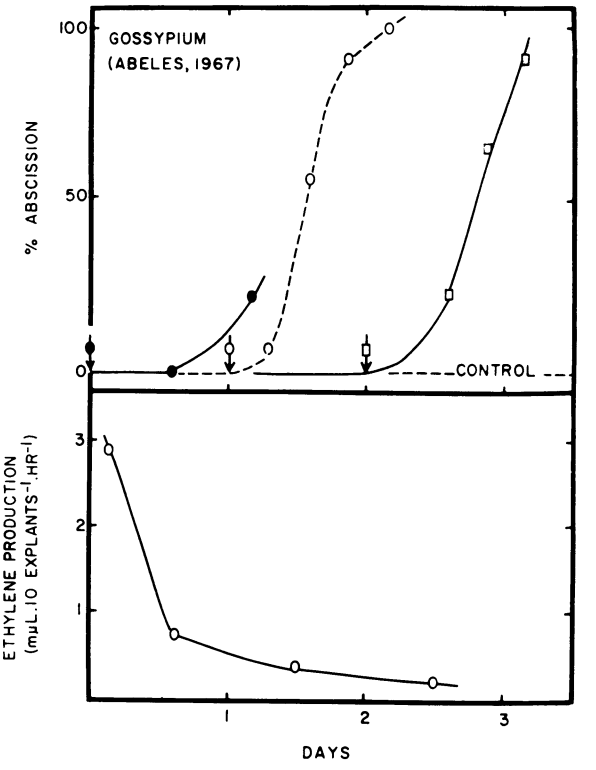

FIG. 4. (Upper) Effect of 0.25 ppm ethylene applied at various times (indicated by arrows) on the abscission of freshly prepared cotton explants (adapted from ref. 2). Control explants did not abscise during the course of the experiment. (Lower) Ethylene production by control explants. A high rate of ethylene production during the first 6 to 12 hours makes the tissue appear to be insensitive to applied ethylene at that time.

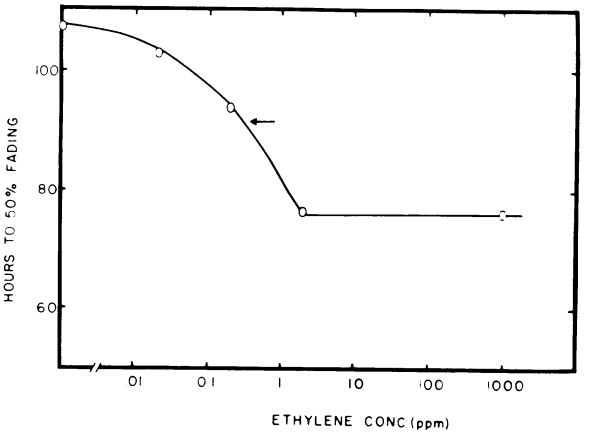

FIG. 5. Fading of 5 day old *Avena sativa* leaves incubated in 10 ml of water in the presence of various concentrations of ethylene. Chlorophyll is measured as the optical density (OD) at 660 $m\mu$ of a 50 ml ethanolic extract obtained from 1 gram of leaves. Arrow indicates half-maximal effect. Data of Dijkman and Burg.

Table II. *Fading of* Avena sativa *Leaves Exposed to 2 ppm Ethylene for Various Periods of Time*

Exposure	OD at 660 mμ per g fr wt after 120 hr[1]
hr	
0	0.59
0-3	0.65
0-6	0.40
0-24	0.40
0-120	0.40

[1] Data of Dijkman and Burg.

Why then does ethylene fail to enhance stage I in explants? Probably this simply reflects the fact that explants initially contain a maximally stimulatory amount of ethylene, making it impossible to demonstrate any response to ethylene applied at that time. During the transition from stage I to stage II the ethylene production decreases, perhaps due to loss of auxin, and consequently the explants become responsive to applied gas.

It has been proposed (5, 26) that auxins prevent abscission by delaying the aging process in the petiole. Of course IAA is not unique in possessing the ability to delay senescence; kinins (61) frequently have this property and GA also may produce the same effect (32). Perhaps these plant hormones are like many animal hormones in this respect, for the latter characteristically function not only to regulate a specific biochemical event but also to *maintain* their target tissue, thus preventing atrophy and death. If senescence must accompany or precede abscission, should not kinins and gibberellins also prevent abscission in at least some cases? Gibberellins accelerate abscission (24, 46) but kinins replace auxin in the bean explant test (2), delaying abscission and preventing ethylene or proximally applied auxin from stimulating abscission. Reversal of ethylene action by kinetin has also been demonstrated in 2 other instances: ethylene inhibits pea bud growth but kinetin overrides the effect (21), and we have noted that *Avena* leaves exposed to ethylene and kinetin remain green much longer than untreated leaves or those induced to fade with ethylene, although not quite as long as those exposed to kinetin alone. Thus in general the facts are consistent with the hypothesis that ethylene accelerates a certain aging process (stage I) which precedes the abscission event. Chatterjee and Leopold (26) and Jacobs (45) have presented evidence which indicates that this aging phenomenon also occurs *in vivo*. Older petioles behave as though they have nearly completed stage I, and in addition have a reduced capacity to be stimulated to abscise when auxin is applied proximally (26). Does ethylene participate in this *in vivo* transition from stage I to stage II? Perhaps it does, for these older petioles in several respects resemble ethylene treated tissue: they have a reduced capacity but a normal velocity of auxin transport (in *Coleus*-Jacobs; Plant Physiol. 43: 1480–95) and a lowered auxin content. If they act like older portions of stems, applied auxins should be only marginally effective in stimulating their ethylene production, and this might explain why it is difficult to stimulate abscission in these petioles by proximal auxin application (see below).

Stimulation of Abscission with GA and Auxins. The extensive studies of Jacobs *et al.* (45) have indicated that correlative events enhancing abscission may be mediated by auxin transported from adjacent or distant leaves and the stem apex to the proximal side of an abscission zone which previously experienced a decline in distally supplied auxin. Pertinent to this problem, but particularly difficult to explain, is the observation of Biggs and Leopold (10) that low concentrations of proximally applied auxin stimulate abscission. This stimulation only develops after the tissue has passed into stage II (25, 63) so clearly the proximal treatment does not prevent the stage I to II transition. Once a petiole has aged and entered stage II it is stimulated to abscise by either distally or proximally supplied auxin (63). Biggs and Leopold (10) believed the stimulation of abscission by low concentrations of proximally applied auxin, and its retardation by high concentrations, to be a reflection of the two phase action which auxin has on many other processes. This explanation is not consistent with recent evidence suggesting that the inhibitory phase of the growth response curve often is due to ethylene formation (17, 21, 24, 42); for on these grounds it would be predicted that high concentrations of auxin ought to accelerate, not inhibit abscission. Abeles (2) has advanced an alternative explanation; he suggests that because of the strict polarity of transport in the petiole a low concentration of auxin applied proximally fails to reach the abscission zone in sufficient quantity to arrest senescence. It does, however, stimulate ethylene production, and this ethylene may in turn enhance abscission after the transition from stage I to II is completed. This attractive idea is supported by his finding that various treatments which facilitate diffusion of auxin from the proximal end to the abscission zone, such as shortening the petiolar stump, reduce the ability of auxin to stimulate abscission (2). Presumably under these conditions enough auxin arrives in the abscission zone at an early time to prevent the transition from stage I to stage II. A similar explanation for the abscission inducing action of a variety of compounds, including GA and certain amino acids, has also been advanced (2, 6). However, there are several reasons for seriously questioning this explanation of the action of proximally applied auxin. The lowest concentration of auxin capable of stimulating abscission is that which causes a just perceptible increase in ethylene evolution (6, 17, 21), and it is unlikely that so slight an increase in ethylene production could significantly alter the ethylene content of an ab-

scission zone several mm distant. For example ethylene produced in the hook region of etiolated pea plants keeps the hook from opening (35) and yet not enough ethylene reaches the subapical zone a few mm away to cause any perceptible swelling. Similarly ethylene causes an intense inhibition of growth on the lower side of a root during the geotropic response, but only a slight inhibition on the upper side (24); and lateral transport of 10 μM IAA through a pea stem gives rise to ethylene production on the lower side of the stem, completely preventing lateral transport there but not in the upper side (17). All of these examples indicate that ethylene acts very close to its site of production in vegetative tissue. Another serious problem is the finding that the same amount of NAA and 2,4-D is present in the abscission zone and subjacent tissue 4 or 24 hours after application, regardless of whether it is introduced proximally or distally (47, 63); hence there is no correlation between the auxin content of the abscission zone and the resultant response, as this explanation presupposes. Finally, it should be noted that it takes less than 1 μM NAA applied proximally to stimulate abscission, whereas more than 100 μM NAA must be applied *either* proximally or distally to retard the process. Apparently much more auxin is required to prevent abscission than is needed to stimulate ethylene production; therefore an explanation based solely on these 2 factors cannot explain why low concentrations of distally supplied auxin fail to stimulate abscission.

Mechanism of Ethylene Action. How Many Actions Does Ethylene Exert? Ethylene causes a multiplicity of effects, but almost without exception there is a remarkable uniformity in the amount of gas which must be applied to produce a threshold, half-maximal and complete response (table I). Although the concentration dependence curve need not be a measure of the affinity of a regulator for its receptor site, it probably is in this case because it is possible by means of this curve to demonstrate competitive inhibition between ethylene and CO_2 (18). This being the case we can interpret the uniformity in dose response curves to mean that all of these ostensibly different effects are controlled by receptor sites having closely similar affinities for ethylene. In addition, in the case of at least 6 different responses (including explant abscission) various analogues of ethylene have the same relative efficacy in each of the test systems, and in these and all other cases examined, CO_2 is a competitive inhibitor of ethylene action (18). These results suggest that a single receptor site is involved in most actions of the gas, and lacking evidence to the contrary we can make a simplifying assumption that this receptor catalyzes a single initial event. The situation may be likened to that of the phytochrome conversion system, where one basic change leads to a diversity of effects which depend mainly on the nature of the tissue.

Certain properties of the receptor site can be inferred from its molecular specificity. The requirements for ethylene action are similar to those which have been established for metal binding to unsaturated compounds, and in fact it has been reported that the biological activity of the various substituted olefines closely parallels their affinity for silver ion (18). In addition, low concentrations of CO replace ethylene in all its functions, and since the action of CO typically involves metal binding, we have proposed that of ethylene to be predicated on the same effect (18).

What is the nature of the initial event catalyzed by ethylene after it has attached to its metallic receptor? The following theories will be considered briefly: i) enhancement of membrane permeability, ii) interaction with IAA, iii) induction of RNA and protein synthesis, and iv) effects on the plant cell wall.

Enhancement of Membrane Permeability. The idea that ethylene affects cellular permeability probably arose because of the high solubility of the gas in lipid. Carbon monoxide because of its very low dipole also is extremely soluble in lipid; yet low concentrations in the range which mimic ethylene action in plants cause death in humans in a matter of hours not by any action in a lipid phase, but by binding to haemoglobin. Similarly, CO binds to cytochrome oxidase not because of its fat solubility but because of its affinity (K_I) for the receptor, which perhaps by coincidence is the same as its affinity (Km) for the ethylene receptor site (18). Another analogue, vinyl fluoride, is highly effective biologically but infinitely less soluble than ethylene in lipid, being equally soluble in lipid and water. Because of these observations, and the fact that ethylene is a very water soluble gas (it has the same water solubility as CO and is 10 times as soluble as O_2 in water), it is not possible from the physical properties of the gas to determine whether its biological action occurs in a lipid phase or elsewhere in the cell. Claims of enhanced permeability due to ethylene application have been published (7, 38), and the idea supported by reports of a progressive increase in leakage during the ripening of fruits (9, 64, 65), but in the author's opinion there is little evidence to support this point of view. The studies on water permeability (7, 38) were carried out under conditions causing a net flow (non-diffusive) of water along an osmotic pressure gradient, and therefore are likely to reflect changes in solute content and wall plasticity rather than altered water permeability, especially in experiments lasting many hours (7). It has not been possible to repeat certain of these studies (38). Moreover ethylene *does not* enhance permeability when it affects pea tissue (22), and biologically active concentrations of ethylene have no effect on mitochondrial permeability (52), although concentrations in the narcotic range are equally damaging to both animal and plant mitochondria. The data on fruit leakage often does not

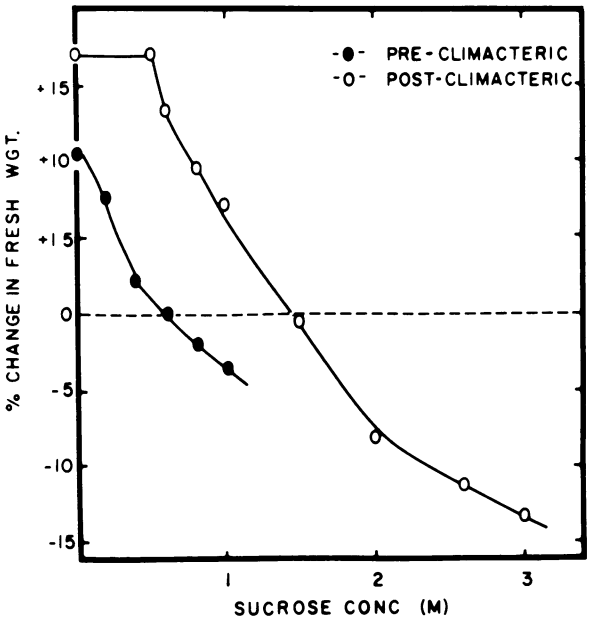

FIG. 6. Fresh weight changes in green and fully ripe Gros Michel banana disks (1 cm diam × 1 mm thick) floated for 60 minutes on solutions containing various concentrations of sucrose. In each case no change in weight occurred when the solution contained a concentration of sucrose having a tonicity closely similar to that of the expressed juice of the fruit. Measurements after 120 minutes, and studies with mannitol (0–0.7 M) gave identical results. The data indicate that at least 40 % of the water contained in the ripe fruit must reside in an osmotic volume.

reflect changes in membrane permeability but rather the total solute available for leakage (22), especially sugar and malate which increase during the climacteric. The concept of 100 % free space in bananas at the time of the climacteric maximum (65) is not compatible with the observation that tissue from climacteric banana fruit has normal osmotic properties (fig 6) and hence a substantial osmotic volume. In any event the reported increase in free space occurs several days before ethylene production begins in this fruit (13) and hence could not be caused by ethylene. Finally it has been demonstrated that certain types of ripe fruit disks most nearly resemble the intact fruit when their integrity is maintained by a solution of moderate tonicity, and under these conditions they do not leak (22). Thus there is little evidence to support the popular view that ethylene may act to alter cellular permeability.

Interaction of Ethylene with IAA. Several effects of ethylene on IAA metabolism have been established although they do not necessarily occur in all types of plant tissue; the gas instantly and reversibly inhibits lateral auxin movement (17), progressively and irreversibly inhibits the capacity of the polar auxin transport system (19, 56, 57); induces IAA oxidase activity (39); apparently retards auxin synthesis (68); and lowers the diffusible auxin level (37, 50, 55, 68). This last mentioned effect, which may result from several of the other actions, probably is a primary factor causing abscission *in vivo*.

Induction of RNA and Protein Synthesis. Enhancement of RNA and protein synthesis after ethylene application has been reported for explants (3, 4, 41) and other tissues (42). It is not clear whether these changes are due to the direct action of the gas, or whether they naturally occur during abscission and simply reflect the fact that abscission has been accelerated by ethylene. It has been reported that at the time of 50 % abscission the rate of incorporation of label into RNA and the type of RNA formed seems to be the same in bean explants regardless of whether abscission has been hastened by ethylene (3). Studies with inhibitors of protein and RNA synthesis cannot resolve this problem because they only indicate whether protein and RNA synthesis are required for abscission and not whether they are needed for the initial steps in ethylene action. It may be significant that in one case, the inhibition of lateral auxin transport by ethylene, it has been shown that cycloheximide fails to prevent ethylene action when it inhibits growth by 50 % (20). This finding implies that ethylene action, at least in this instance, does not require protein synthesis.

Effect of Ethylene on the Cell Wall. Ethylene causes growing cells in most roots and many stems immediately to reduce their rate of elongation and expand instead in a radial direction (17, 21, 24). As the normal predominantly longitudinal direction of expansion is thought to be imposed on cells by the restricting influence of radially deposited microfibrils, this observation suggests that ethylene somehow alters the structure of the cell wall to allow radial expansion. Ethylene also is highly effective in stimulating root hair development (24), possibly explaining why high concentrations of auxin have the same action; this again implies an effect of ethylene on the cell wall. The rate of incorporation of ^{14}C-glucose into pea cell walls is slightly reduced by ethylene (11), but most of the effect can be ascribed to a small decrease in the rate of glucose uptake. When viewed through crossed polaroids, the swollen cells display a characteristic pattern of longitudinal banding in the walls (21) which is indistinguishable from that observed in the same cells induced to swell by benzimidazole (60). In the latter case the banding is due to newly deposited longitudinally oriented microfibrils, so presumably these also are formed in ethylene treated cells.

Changes in cell wall metabolism caused by ethylene are likely to have significance for the abscission process since dissolution of walls and the middle lamella are an important aspect of it. During abscission cellulase increases in the abscission zone (43), and this change is prevented by IAA and stimulated by ethylene. To the contrary, IAA *in-*

duces cellulase activity in the subapical zone of peas when it causes swelling (31), but it should be noted that in this experiment the concentration of IAA used was so high that it undoubtedly stimulated extensive ethylene evolution, and it is clear that this ethylene and not the applied IAA causes the swelling in peas (17). The exact role which cellulase might play in the swelling reaction is not obvious since the total wall dry weight and rate of increase in wall dry weight is not altered by ethylene applied to pea stem sections (17), but in the case of abscission a hydrolytic process could be extremely important.

Evidence has been reviewed which suggests an involvement of ethylene in the control of senescence and abscission. Applied gas probably acts to lower the level of diffusible auxin by inhibiting auxin synthesis and transport, and enhancing auxin destruction. Subsequently ethylene may accelerate senescence and eventually abscission itself, possibly through its action on plant cell walls.

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