Relationship of Pectic Enzymes to Abscission

Daryl D. Smith

University of Northern Iowa
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DARYL D. SMITH

Abstract. A comprehensive review of work pertinent to changes in the separation zone prior to abscission is presented. Since these changes are largely pectic in nature the involvement of pectic enzymes is suggested. The results obtained for the role of pectin methylesterase (PME) in abscission are as follows: (1) A localization of PME activity in the abscission zone of tobacco pedicels was established; (2) Pollination of tobacco flowers is followed by an increase in PME activity in the abscission zone while PME activity remains static after prevention of pollination (the flower abscises within a week if not pollinated); (3) Application of indole acetic acid to unfertilized tobacco ovaries stimulated an increase in PME activity of the abscission zone of the pedicel.

Early work with abscission was primarily anatomical although there was some speculation concerning the regulation of abscission. Von Mohl (1860) noted a definite separation layer in which abscission always occurred. Within this layer cells separated from one another with their walls still intact. Wiesner (1871) confirmed some of von Mohl's observations and theorized that the dissolution of the intercellular substances of the separation layer was caused by organic acids developed in the leaves. Molisch (1886) suggested that a gum ferment might be the cause of the dissolution process. Kubart (1906) concurred with Wiesner (1905) that an organic acid was responsible for the dissolution of the middle lamella, but he admitted the possibility of enzymatic involvement in the abscission process.

At the turn of the century a conflict developed concerning the manner in which abscission occurs. Tison (1900) reported dissolution of the primary cell walls of the separation layer in addition to the middle lamella. This was confirmed by later workers (Lloyd 1914, Dutt 1928). However, Lee (1911) concurred with von Mohl (1860) and reported that abscission of leaves involved a dissolution of the middle lamella between the primary walls of adjacent layers. This type of abscission was also noted in several types of flowers by Hannig (1913). In addition, Hannig (1913) and Myers (1940) observed disintegration of cells during abscission. More recently this disagreement was still evident as Yager (1957) noted dissolution of the middle lamella while Lineweber and Hall (1959) reported hydrolysis of the primary wall as well as the middle lamella. Addicott and Lynch (1951) concluded that the critical

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2 Department of Biology, University of Northern Iowa. The assistance of Dr. Robert Muir, Department of Botany, University of Iowa is gratefully acknowledged.
phase of leaf abscission was the dissolution of the entire cell. However, Addicott (1965) modified this somewhat by stating that in some cases little more than the middle lamella was digested. Rubinstein and Leopold (1964) expressed the opinion that anatomical changes during abscission vary somewhat from plant to plant. Species variation in the manner in which abscission occurs had been noted in 1928 by Pfeiffer. A recent extensive examination of the anatomical aspects of abscission by Webster (1968) coupled with the electron microscopy work of Morre (1968) permit a more definitive view of the subject. Apparently local breakdown of intercellular materials between adjacent cells of the separation layer is initiated in the middle lamella. However, subsequent dissolution of cell walls and digestion of cellular content may occur. Morre (1968) is of the opinion that dissolution of the intercellular cement is necessary, and perhaps sufficient, for formation of the separation layer during abscission.

Knowledge of changes in the middle lamella that occur during abscission would be quite useful in an elucidation of the abscission process. The pectinaceous nature of the middle lamella has been established for some time (Mangin 1889). In fact, Mangin's work served as a basis for some workers referring to the abscission process as a dissolution of the protopectin and calcium pectate of the middle lamella. Lloyd (1916) spoke of the dissolution of the middle lamella as a process of hydrolysis and apparently assumed that enzymes were involved. As early as 1918 Kendall reported the appearance of pectin as abscission occurred in tobacco flowers and assumed that enzymes were involved in the dissolution process. Sampson (1918) determined that the final stage of changes in the middle lamella prior to cell separation was the breakdown of calcium pectate. Facey (1950) using histochemical procedures in the study of the abscission zone of Fraxinus cuttings demonstrated a shift from calcium pectate to pectic acid and then to water soluble pectin. She suggested that esterification of pectic acid to the form of pectin does occur during abscission, since pectin is the only form that is water soluble. Rasmussen (1965) and Morre (1968) reported similar results. Morre considered that the observed increase in solubility of pectins of the middle lamella might arise from removal of polyvalent cations, methylation of carboxyl groups and/or depolymerization. The appearance of pectin lends support to the idea of methylation of carboxyl groups. However, in vitro evidence exists (McClendon 1964, Yager 1960, Zaitlin and Coltrin 1964) to support the thought that depolymerization of pectic substances or removal of ions will stimulate natural separation. That esterification may be involved in abscission has been emphasized by the work of Yager and Muir (1958a, b). They observed that methionine, a methyl donor, caused a very rapid acceleration of abscission of un-
fertilized ovaries of tobacco. Methionine and certain other amino acids have been found to contribute methyl groups for esterification of pectin and other substances of the abscission zone by Nelson (1960) and Valdovinos and Muir (1965).

Recently work concerning enzymes involved in abscission has focused on pectic enzymes. However, various other enzymes have been reported to be associated with the abscission process. Sampson (1918) found oxidases to be present in all tissue of the abscission zone except the xylem; however in the stem and petiole, oxidases were present only in epidermal and phloem tissue. In 1919 Heinicke noted that catalase was more active in the abscission zone than in contiguous areas. Kertesz (1943) suggested that peroxidase may act to change pectic compounds in plant tissue. Carns (1951) applied inhibitors of specific respiratory enzymes to explants and found that abscission was retarded. Cellulase has been specifically associated with wall changes in the abscission zone (Horton and Osborne 1967).

Since abscission is initiated in the middle lamella with the dissolution of pectinaceous material, the action of pectic enzymes should be relevant. However, there is a general lack of information concerning pectic enzymes other than pectin methylesterase (PME). Much of the work with pectic enzymes has been done in relation to fruit ripening (Bateman and Millar 1966, Demain and Phaff 1957, Kertesz 1951, Lineweaver and Jansen 1951, Sterling and Kalb 1959), a process which may be analagous to the abscission process.

Bonner (1936), speculating from the work of Sampson (1918), suggested that protopectinase was responsible for leaf abscission although its existence has been debated. Pectin-polygalacturonase is probably the best known of the pectic enzymes other than PME. Demain and Phaff (1954, 1957) reported a complex of enzymes instead of a single pectin-polygalacturonase. The complex consisted of several exo-polygalacturonases specific for pectins of different degrees of esterification and size of polymer and a single endo-galacturonase. Schubert (1952), Dingle et al. (1953) and Ayres et al. (1952) indicated that the endo-polygalacturonase is a complex of enzymes. Pectinase is frequently used to describe pectin-polygalacturonase and is also used to designate pectic enzyme mixtures. The complexity in the identification of specific pectin-polygalacturonases may explain some of the conflicting reports as well as the problems arising from nomenclature. McClendon and Somers (1956) reported the occurrence of two pectin-glycosidases with different conditions required for activity. Kertesz (1951) postulated the probable occurrence of additional pectic enzymes in higher plants. Sato, Byerrum, and Ball (1957) reported the bio-
synthesis of methyl esters of pectinic acid through transmethylation from methionine with a preparation of radish tissue. Albersheim, Neudom, and Deuel (1960) isolated a hydrolytic enzyme that acts as a transeliminase by splitting methylated pectin. Albersheim and Killias (1962) established the presence of the enzyme in higher plants (pea) and Albersheim (1963) found that it could be inhibited by 2,4-D and indole acetic acid (IAA). Yager (1960) reported the in vitro effects of pectinase and a pectin glycosidase on tobacco tissue. These two enzymes caused dissolution of the middle lamella and separation of cells of the abscission zone as well as other cells of the tissue slice through the pedicel. Methionine and low concentrations of IAA increased the dissolution activity of the two enzymes while high concentrations of IAA retarded their activity. Morre (1968) found that pectinase activity which was low or absent at the time of excision, rose to a maximum at 72 hours and then declined. Appearance of activity coincided closely with separation layer formation and declined when separation was complete. The pectinase of the bean explant was assumed to be an endopolygalacturonase.

Pectin methylesterase (PME) has been studied more extensively than the other pectic enzymes. It is widely distributed in higher plants and appears to be associated with cell walls. Its action is generally considered to be that of catalyzing the hydrolysis of the methyl ester bonds of pectinic acid and pectin (Kertesz 1951). Lineweaver and Jansen (1951) suggest that PME may act only on ester bonds that are adjacent to free carboxyl groups or may act on these more rapidly than on other ester bonds. Much of the work has been in relation to plant growth (Bryan and Newcomb 1954; Glasziou 1959 and Jansen, Jang and Bonner 1960). Osborne (1958) found that PME activity decreases with the age of the bean leaf. The greatest drop in activity was found in pulvinus tissue as compared with other parts of the blade or petiole. Application of ethylene accelerated abscission while application of such compounds as 2,4-D sustained activity of the enzyme. Yager (1960) found PME activity to be high in the abscission region of tobacco pedicels. In addition, high concentrations of IAA which delayed abscission, increased PME activity and methionine, an accelerator of abscission, decreased PME activity. LaMotte, etc. (1960) compared PME activity in various portions of attached leaves and abscissing petioles and confirmed some of the earlier findings of Osborne (1958) and Yager (1960). The team found that PME activity in both Coleus and bean plants was lower in the distal portion of abscission zones of abscissing petioles than in attached leaves. Also, auxin treatment of debladed petioles of Coleus prevented abscission and resulted in small increases in PME activity in abscission zones and most of the other regions sampled.
The above relationship of the activity of pectic enzymes to auxins coupled with the effect of auxins on abscission provides fuel for some interesting speculation concerning the physiological control of abscission. Many workers have found evidence that an endogenous regulator is responsible for controlling abscission. Among the first was Laibach (1933) who reported orchid pollen (a rich source of auxin) could effectively delay abscission of debladed Coleus petioles. This observation was confirmed by LaRue (1936) who found that the retarding effect of pure indoleacetic acid compared with the intact leaf. Myers (1940) related the amount of auxin in a leaf blade to its inhibition of abscission. Wetmore and Jacobs (1953) found a similar correlation between the normal longevity of intact leaves and their content of diffusible auxin. Since auxins affect abscission and pectic enzymes are associated with abscission the question arises as to how they are related. Osborne (1958) found that 2, 4-D sustained activity of PME. Yager (1960) observed the following in vitro effect of varying IAA concentration: low concentrations of IAA, which accelerated abscission, caused a decrease in PME activity of tissue macerates and an increase in action of pectinase and pectin-glycosidase whereas higher concentrations of IAA, which retarded abscission, increased PME activity but inhibited pectinase and pectin-glycosidase. This correlates well the work of Gaur and Leopold (1955) and Biggs and Leopold (1958) who found that dilute concentrations of auxin accelerated abscission, whereas higher concentrations were inhibitory. Investigators using other types of plant tissue have observed auxin induced increases in PME activity (Neely, etc. 1950, Bryan and Newcomb 1954).

The dual abilities of auxin either to inhibit or promote abscission somewhat complicate the issue. Rubinstein and Leopold (1964) suggest the auxin effects may operate through mechanisms involving changes in membrane permeability, changes in pectic enzyme activities, or changes in the production of ethylene by the petiole tissue.

The following is a report of the results of a series of investigations in an attempt to evaluate the role of PME in abscission of tobacco flowers, Nicotiana tabacum L. cultivar Lizard's Tail. The relative activity of the enzyme was determined by an increase in free carboxyl groups as suggested originally by Kertesz (1937). The results are reported in three parts: (1) distribution of PME in the tobacco pedicel; (2) comparison of PME in the abscission zones of pollinated and unpollinated flowers; (3) effect of IAA treatment of tobacco ovaries on the PME activity of the abscission zones of tobacco pedicels.

The localization of PME activity in the pedicels of tobacco flowers was examined by comparing tissue of the abscission zone to
tissue of similar weight from the region of the pedicels just below the receptacle. It is evident from the data of Table 1 that the PME activity is higher in the abscission zone of the pedicel than it is in the distal region. This confirms the earlier work of Yager (1960). As can be seen there is considerable variation in the PME activity of the abscission zones from pedicels of flowers at anthesis.

### TABLE 1

Distribution of Pectin Methylesterase Activity in the Tobacco Pedicel

<table>
<thead>
<tr>
<th>Trial</th>
<th>Days After Pollination</th>
<th>Abscission Zone</th>
<th>Apical Region</th>
<th>Abscission Minus Apical</th>
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<tr>
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<td></td>
<td>2</td>
<td>8.87</td>
<td>4.59</td>
<td>4.28</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>11.13</td>
<td>4.12</td>
<td>7.01</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>11.19</td>
<td>6.60</td>
<td>4.59</td>
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<td>2</td>
<td>11.78</td>
<td>8.77</td>
<td>3.01</td>
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<tr>
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<td>4</td>
<td>11.41</td>
<td>8.09</td>
<td>3.32</td>
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<td>8.09</td>
<td>4.43</td>
<td>3.66</td>
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<td>3.41</td>
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<tr>
<td></td>
<td>3</td>
<td>7.41</td>
<td>—</td>
<td>—</td>
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<td>7.91</td>
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<td>2.36</td>
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<tr>
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<td></td>
<td>14</td>
<td>11.56</td>
<td>7.84</td>
<td>3.72</td>
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<tr>
<td>6</td>
<td>10</td>
<td>12.80</td>
<td>5.49</td>
<td>7.31</td>
</tr>
</tbody>
</table>

Following pollination of the tobacco flower an increase in the PME activity is observed in both the abscission zone and distal region of the pedicel. However, the higher activity in the abscission zone is maintained over that of the distal region of the pedicel. The differences in activity between the two regions indicates a tendency toward a slight increase in the difference following pollination. This suggests that the PME of the abscission zone is more responsive to changes in the ovary following pollination. Pollination and fertilization initiate growth and development of the ovary with a corresponding decline in the possibility of abscission; perhaps the higher PME activity could account for this. Higher PME activity would catalyze more rapid deesterification thus preventing formation of water soluble pectin and retarding separation in the abscission zone. The localization of a larger amount of enzyme in the
TABLE 2
Effect of Pollination on Pectin Methylesterase Activity

<table>
<thead>
<tr>
<th>Days After Pollination</th>
<th>Relative PME Activity (mg CH₃O/gm fresh weight/30 min)</th>
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<tbody>
<tr>
<td></td>
<td>0</td>
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<tr>
<td></td>
<td>3.29</td>
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<tr>
<td></td>
<td>6.70</td>
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</table>

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abscission zone suggests the possibility of a primary role in the abscission process.

If pollination and fertilization do not occur, the flowers of *Nicotiana* wither and abscission occurs at the base of the pedicel within four to eight days after anthesis. With successful pollination, growth of the ovary is initiated and development of a fruit begins. A number of experiments were performed to determine if the PME activity in the abscission zone of the tobacco pedicel was affected by pollination. The PME activity in abscission zones of pedicels from pollinated and unpollinated flowers is recorded in Tables 2 and 3. There is day to day variation in PME activity and

### TABLE 3

Effect of Prevention of Pollination on Pectin Methylesterase Activity

<table>
<thead>
<tr>
<th>Days After Prevention</th>
<th>Relative PME Activity (mg CH₂O/gm fresh weight/30 min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>3.28</td>
</tr>
<tr>
<td>Date of Analysis</td>
<td>Dec 22</td>
</tr>
</tbody>
</table>

comparison of values from different days must be tentative. As noted previously in Table 1 this variation resulted in a considerable range of PME activity in tissue from the abscission zone of tobacco pedicels. This variation is undoubtedly a reflection of an environment influence.

The data obtained on May 6, and May 12, is presented in Figure 1. This affords a more direct comparison of the effect of pollination and lack of pollination upon PME activity. There is a slight increase in PME activity during the first two days after pollination, but the major increase in activity is from the second to the fourth day. This major increase in activity occurs at the same time as rapid elongation of the ovary as observed in Figure 2. A decline in activity between the third and fifth day is also noted in determination of March 3. This decline is not evident in all determinations, indicating that this is not a consistent trend, but
Fig. 1. Pectin methylesterase activity in abscission zones of tobacco pedicels after pollination or prevention of pollination.

- - - - pollinated

- - - - unpollinated

Fig. 2. Elongation of tobacco overay after pollination and fertilization.
fluctuation in PME activity. Generally speaking, an increase in PME activity follows pollination. There is a very little change in PME activity in the abscission zone of unpollinated flower pedicels. On May 12, the activity declined the first day after pollination had been prevented, but returned to the level of anthesis on the second day. The third day is somewhat higher than anthesis. By the fifth day after prevention of pollination the PME activity had again dropped slightly below the value at anthesis. Apparently the PME activity fluctuates after pollination is prevented, but the overall change is not very great. Usually the majority of unpollinated flowers abscise within one week after anthesis or shortly thereafter.

It is interesting to note that changes in PME activity in the abscission zones correspond quite closely with changes in diffusible auxin content of the ovary and pedicel as determined by Muir (1942). His data is presented in Fig. 3. He noted a marked increase in diffusible auxin at the time the pollen tubes reached the ovary (approximately 25-40 hours after pollination). The diffusible auxin of the pedicel did not begin to increase until 45 hours after pollination.

![Graph](https://scholarworks.uni.edu/pias/vol76/iss1/8)

**Fig. 3.** Diffusible auxin obtained from the ovaries and pedicels of pollinated and unpollinated tobacco flowers (data of Muir 1942).

- PO: pollinated ovary
- PP: pollinated pedicel
- UO: unpollinated ovary
- Up: unpollinated pedicel
pollination and increased rapidly from about 48 hours to 65 hours after pollination. Lund (1956) measured changes in extractable auxin content of the style and ovary of tobacco flowers following pollination. He also noted that the auxin content of the ovary begins to rise at about 50 hours after pollination at which time the ovary begins to enlarge (Fig. 2). Changes in extractable auxin of the ovary as measured by Lund (1956) were not as sharply delineated as the measurement of diffusible auxin by Muir (1942). Also, changes in PME activity most closely parallel changes in diffusible auxin. This suggests that diffusible auxin is the physiologically active auxin affecting ovary growth and PME activity. This could indicate that the auxin is involved in enzyme synthesis or that a certain period of time is required for activation of PME by auxin. However, the parallel development of the two is strongly indicative of a relationship. Evidently some fluctuation of PME activity in the pollinated flower is shown by the drop in activity from the fourth to the fifth day. Muir (1942) reported almost the same sort of drop in diffusible auxin from 60-80 hours in one of his determinations (Fig. 3). His other determinations over the same time interval did not show this drop. A drop in PME activity was observed in three of nine trials. The level of PME activity or diffusible auxin never did drop to that of unpollinated flowers. Possibly these fluctuations are related to a decrease in the rate of growth of the tobacco ovary shown in Fig. 2.

The identification by Lund (1956) of IAA as one of the principal forms causing increase of extractable auxin in tobacco ovaries relates very well to the work of Yager (1960) on the effect of varying concentrations of IAA on PME activity. Yager reported that addition of 5 ppm IAA to an assay solution containing tissue macerate of the abscission zone from tobacco pedicels decreased PME activity. Additions of 100 and 1000 ppm IAA increased PME activity. This suggests that production of IAA by pollinated flowers is sufficient to induce greater PME activity. Conversely, the IAA of the unpollinated flower is either not present in sufficient quantities to affect the PME activity in the abscission zone, or there is just enough present to decrease PME activity. A decrease in PME activity would permit esterification of the pectinic acid resulting in the formation of water soluble pectin. Following the formation of pectin the middle lamella would dissolve and abscission could occur. On the other hand, the production of IAA in the ovary of the pollinated tobacco flower could produce IAA in sufficient quantities to cause an increase in PME activity. With increased PME activity there would be a corresponding increase in hydrolysis of methyl esters of pectin. This would result in the maintenance of the cementing substance of the middle lamella,
calcium pectate, and abscission would be prevented. Yager (1957) reported a slight acceleration of abscission of tobacco ovaries with treatment of 5 ppm IAA. However, this acceleration was not consistent since in another instance abscission was retarded slightly. Treatment of tobacco varies with very minute concentrations of IAA would be helpful in evaluating this situation.

A more direct examination of the effect of IAA on PME activity of the abscission zone was conducted. A $10^{-2}$M solution of IAA was injected into the tobacco ovary under pressure until the solution flowed from the cut tips of the sepal. This method of treatment insured that the solution was perfused throughout the open space of the ovary.

The analysis of the effect of IAA on PME activity in the pedicel of tobacco is shown in Table 4. Parthenocarpic development of the

<table>
<thead>
<tr>
<th>Days After Treatment</th>
<th>Relative PME Activity (mg CH$_2$O/gm fresh weight/30 min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>4.99 7.63 7.75</td>
</tr>
<tr>
<td>1</td>
<td>—      —    9.42</td>
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<td>2</td>
<td>—      —    12.40</td>
</tr>
<tr>
<td>4</td>
<td>7.87 10.78 —</td>
</tr>
<tr>
<td>6</td>
<td>—      10.51 —</td>
</tr>
<tr>
<td>Date of Analysis</td>
<td>Feb 4   Feb 11 Feb 23</td>
</tr>
</tbody>
</table>

The data indicates that IAA causes an increase in PME activity. As can be seen in Fig. 4 the PME activity rose rapidly following treatment with IAA reaching a peak of activity twice that of the PME activity at anthesis. A few of the IAA-treated ovaries abscised, but no more than would be expected from ineffective application of the solution. After ten days there was some abscission of the IAA-treated ovaries, but at the end of two weeks a considerable number remained on the parent plant.

The total length of tobacco pedicels and total ovary length following treatment with IAA is given in Fig. 5. The observations provide further inferences concerning the relationship of diffusible auxin and PME activity. The increase in length of the tobacco ovary following IAA treatment is almost identical to the increase in length of the pollinated ovary during the first five days after pollination. However, maximum length attained by the IAA-treated ovaries is 20 mm as compared with 25 mm for pollinated ovaries
Fig. 4. Relative pectin methylesterase activity in abscission zones of tobacco pedicels following treatment of unfertilized ovaries with indoleacetic acid (IAA).

Fig. 5. Total length of pedicel and total length of ovary after treatment of unfertilized ovaries of tobacco with indoleacetic acid (IAA).
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(Fig. 2). The rapid growth of the IAA-treated ovary indicates that considerable auxin is present. This amount of auxin may be super-optimal for pedicel growth and therefore would inhibit elongation. This high amount of auxin would stimulate high PME activity, as observed by Yager (1960), and prevent abscission of the ovary.

Apparently IAA is acting to stimulate PME activity, and thus, prevent dissolution of the middle lamella. The response of increased PME activity was more rapid following IAA injection than after pollination. This would be expected since injection of IAA into the ovary permits more rapid movement through the pedicel into the abscission zone than would result from naturally produced auxin resulting from pollination and fertilization. A decline in PME activity a few days after injection suggests that the natural supply of auxin may be more consistent. However, injection of IAA induces parthenocarpic development of the ovary and this “ovary” can then supply sufficient auxin to the abscission zone to maintain high PME activity and thereby prevent abscission.

In summation, the evidence suggests that PME could control abscission by catalyzing deesterification of pectic substances to maintain the cementing substance of the middle lamella, calcium pectate. Pectin methylesterase activity is enhanced by high concentrations of auxin and abscission is retarded. Prior to abscission the production of auxin decreases. This low supply of auxin could result in decreased PME activity and/or increased activity of pectin-polygalacturonase. Another possibility is that with declining auxin other natural stimulators of abscission, e.g. amino acids, could predominate and promote esterification, via a transmethylase, of the pectic compounds. Once methylation has occurred other enzymes such as pectin transeliminase or pectin-polygalacturonase could act to further hydrolyze the pectin of the middle lamella.

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