The Effect of Indole-3-acetic Acid and Other Growth Regulators on the Ripening of Avocado Fruits

PETER O. TINGWA* AND ROY E. YOUNG
Department of Plant Sciences, University of California, Riverside, California 92502

ABSTRACT

Observations were made of the effects of several plant regulators, indole-3-acetic acid, kinetin, abscisic acid, and gibberellin, as well as of extracts prepared from leaves and fruit stalks on the respiration pattern, ethylene production, and the number of days to ripen of avocado fruits (Persea americana Mill.). These substances were vacuum infiltrated to insure good penetration and distribution. Kinetin, abscisic acid, gibberellin acid, and the extracts had no effect on either ripening time or on the respiration pattern and ethylene production of the fruits. Indoleacetic acid, however, had a marked effect on ripening. At high concentrations (100 and 1000 µM), indoleacetic acid stimulated respiration and induced preclimacteric ethylene production, resulting in accelerated ripening of the fruits. At the low concentrations (1 and 10 µM), it delayed ripening of fruits and suppressed the climacteric respiration and ethylene production. The results reinforce several previous observations with other fruits that auxins may largely constitute 'resistance to ripening' and may be responsible for the lack of ripening shown by unpicked fruits.

The inability of some fruits, notably the avocado, to ripen on the tree (3, 7, 8), coupled with the lack of response by freshly harvested fruits to ethylene treatment (16, 20, 23), have prompted several workers to propose the existence of a ripening inhibitor in attached fruits (3, 7, 8). This inhibitor is presumed to be formed in leaves of the parent tree and translocated to the fruit while still attached (3, 7, 8). Following harvest, the inhibitor is inactivated during the preclimacteric period and ripening is initiated by endogenously produced ethylene (14, 16, 20, 23). Plant hormones have been shown to affect some aspects of fruit ripening. For example, indoleacetic acid has been reported to delay the onset of the climacteric in bananas (30) and pears (14), and both gibberellins and cytokinins have been shown to delay the degradation of Chl in ripening tomatoes (1) and orange peel (12).

A growing body of information indicates that the ripening inhibitor may be an auxin (14, 15, 27). Direct evidence that auxins constitute the resistance to ripening was demonstrated by the delay of ripening processes in bananas (30) and pears (14) following treatments with either 2,4-dichlorophenoxy-
2a). The cause of the burst of CO₂ production is presumed to be attributable to either an injury effect, or a sudden increased availability of O₂ to an accumulated oxidizable substrate. Although the practice of vacuum infiltration enhanced preclimacteric respiration, it caused the opposite effect for climacteric respiration.

**IAA Effect.** The influence of increasing concentration of IAA from 1 to 1000 μM on the respiration pattern of ‘Fuerte’ and ‘Hass’ fruits is shown in Figures 1a and 2a, respectively. When compared to water-infiltrated controls, preclimacteric respiration was stimulated by IAA and the magnitude of stimulation was proportional to its concentration. The reduced respiration rate at the climacteric resulting from the practice of infiltration was reversed by higher IAA concentrations (100 and 1000 μM), such that the climacteric respiration in these treatments was as high as that of uninfiltrated controls. On the other hand, low IAA concentrations (1 and 10 μM), while stimulating preclimacteric respiration, depressed the climacteric respiration rate even further.

The effect of IAA on ethylene production is shown in Figures 1b and 2b for ‘Fuerte’ and ‘Hass’ varieties, respectively. High concentrations of IAA induced preclimacteric ethylene production earlier and stimulated its rate of evolution during the climacteric. The peak production of ethylene by fruits given these higher IAA treatments came before those of the controls. Low IAA levels (1 and 10 μM) caused a delay in initiation of ethylene production during the preclimacteric period and both delayed and reduced its production at the climacteric peak.

![Figure 1](image1.png)

**Fig. 1.** Effect of increasing concentration of IAA on respiratory pattern (a) and ethylene production (b) of ‘Fuerte’ avocado fruits at 20 C. The IAA concentrations were 1 μM (△), 10 μM (○), 100 μM (□), 1000 μM (●), and uninfiltrated (○) and water-infiltrated (●) controls. Each point represents the average of four fruits.

![Figure 2a](image2a.png)

**Fig. 2.** Respiration pattern (a) and ethylene production (b) of ‘Hass’ avocado fruits vacuum infiltrated with water (●), 1 μM (△), 5 μM (■), and 10 μM (▲) IAA at 20 C. Uninfiltrated fruits are presented as (○). Each point is the average of four fruits.

When judging the effect of IAA (or any of the other treatments) on the time of ripening, the days preceding the climacteric ethylene peak were considered to be the number of days taken to ripen because this point is related to ripening and may be determined with great accuracy. Therefore, as shown in Figures 1b and 2b, low concentrations of IAA delayed ripening of fruits by as much as 2 to 3 days at 20 C whereas high concentrations accelerated ripening. This delay of ripening induced by low IAA concentrations was much more pronounced in the midseason ‘Fuerte’ (Fig. 1b) than in late season ‘Hass’ fruits (Fig. 2b).

**Kinetin, GA₃, ABA, and Extracts.** The respiration pattern and ethylene production of fruits infiltrated with 1 or 10 μM solutions of GA₃, kinetin, ABA, or with 1:4 and 1:8 dilutions of leaf or peduncle extracts did not differ from uninfiltrated or water-infiltrated controls. There was, however, some preclimacteric stimulation of ethylene production by fruits infiltrated with 10 μM kinetin.

The effects of these treatments on ripening time are given in Table I. Although there was some variability in the ripening time of individual fruits, none of the concentrations of these treatments affected the time of ripening. In contrast, the ripening time of fruits infiltrated with 1 μM IAA was uniform and delayed by 2.5 days when compared to the controls.

Results of further investigations with half fruits showed no significant difference between ripening time of halves treated with 10 μM ABA or 1:4 peduncle extract and their corresponding water-treated halves. In each case, the respiration pattern and ethylene production were similar in both halves of the
fruit as shown in Figure 3, a and c. In contrast, whenever one half of a fruit was treated with 1 μM IAA, its respiration and ethylene production were suppressed and its ripening was delayed (Fig. 3b).

DISCUSSION

These experiments show that IAA exerts a marked effect on the ripening of detached avocado fruits, thus providing the first indications of the involvement of auxins (IAA) in the ripening of avocado fruits. They support previous reports for pears (14) and banana slices (30) that auxins can delay the ripening of fruits, provided adequate penetration of the substance is insured by infiltration. Opposite effects, resulting from poor penetration of auxins, have been reported (5, 19) and alluded to (14, 30) in pears and bananas.

Treatment of avocado fruit with IAA seems to exert control over the time of induction of the climacteric as well as over the amount of ethylene produced during the climacteric.

Table I. Effect of Various Hormones and Leaf and Peduncle Extracts on the Ripening Time of ‘Hass’ Avocado Fruits at 20 C

<table>
<thead>
<tr>
<th>Substance Infiltrated</th>
<th>Days to Ripen</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>6.8</td>
</tr>
<tr>
<td>Water</td>
<td>6.7</td>
</tr>
<tr>
<td>1 μM IAA</td>
<td>6.1</td>
</tr>
<tr>
<td>1 μM Kinetin</td>
<td>7.0</td>
</tr>
<tr>
<td>10 μM Kinetin</td>
<td>6.8</td>
</tr>
<tr>
<td>1 μM GA3</td>
<td>7.0</td>
</tr>
<tr>
<td>10 μM GA3</td>
<td>6.8</td>
</tr>
<tr>
<td>1 μM ABA</td>
<td>6.8</td>
</tr>
<tr>
<td>10 μM ABA</td>
<td>6.6</td>
</tr>
<tr>
<td>1:8 Leaf extract</td>
<td>7.1</td>
</tr>
<tr>
<td>1:4 Leaf extract</td>
<td>6.7</td>
</tr>
<tr>
<td>1:8 Peduncle extract</td>
<td>6.9</td>
</tr>
<tr>
<td>1:4 Peduncle extract</td>
<td>7.4</td>
</tr>
</tbody>
</table>

Concentrations of IAA above 10 μM caused more rapid ripening and higher ethylene production, an effect reported for auxin treatments in other fruits as well (9, 14, 19, 30). IAA concentrations less than 10 μM caused delayed ripening and ethylene production lower than the controls. These seemingly contradictory observations suggest that IAA is acting on two different systems. Dual effects of auxin have been proposed in other cases, e.g., pea root tips (28) in which IAA affected two systems influencing ethylene synthesis; the low concentration treatment was shown to act directly on an enzyme whereas the high concentration system required protein synthesis. Likewise in leaf abscission experiments, IAA plays a dual role in that, in general, auxin promotes abscission at low treatment levels and inhibits at higher levels (2). In the case of IAA effects on stem elongation, stimulation at low and inhibition at high levels is the rule (29). Vendrell (30) has suggested that auxins affect two regulatory systems in bananas; one concerned with regulation of respiration and ethylene production and the other with desensitizing fruits to the effects of ethylene.

We propose a dual effect for IAA in avocado ripening. The high concentration effect is to induce early ethylene production which in turn initiates ripening. It may be noted in Figure 1 that the first increase in ethylene production precedes the increase in respiration by a few hours when 100 μM IAA was used. On the other hand, the first increase in ethylene production in the 1 μM-treated fruit and in the control fruit occurred about 12 hr after respiration increased. We suggest that treatment with 1000 μM IAA caused increased and earlier ethylene production, which initiated ripening just as does treatment with ethylene after picking (4, 16). Ethylene production in response to auxin treatment has been reported for a number of plant tissues (24).

The low concentration effect of IAA is clearly at a different site because ethylene production was both delayed and diminished, relative to the control (Fig. 1). Assuming that the low IAA concentration effect mimics the process by which avocados remain unripe on the tree, different mechanisms may be visualized for the effect of IAA, depending on which of two alternatives are assumed: (a) a ripening inhibitor is translocated from tree to fruit; or (b) a ripening hormone produced in the fruit is kept at a low level by movement into the

---

Fig. 3. Respiration pattern and ethylene production of 'Fuerte' half fruits infiltrated with (a): 10 μM ABA (), (b): 1 μM IAA (■), and (c): 1:4 peduncle extract (▲), all at 20 C. The other halves of the same fruits infiltrated with water are shown as (○).

---

Downloaded from on July 21, 2017 - Published by www.plantphysiol.org
Copyright © 1975 American Society of Plant Biologists. All rights reserved.
branch before detachment and ripening is initiated by accumulation of the hormone after detachment. If ripening in attached fruit is prevented by the first alternative and ripening is initiated by depletion of IAA, treatment of the detached fruit with low levels of IAA would contribute to the endogenous pool and extend the preclimacteric period. This is so long as the IAA concentration is below the level that induces ethylene production. If the second alternative is the case, i.e., ripening initiated by a hormone accumulating in detached fruit, low concentrations of IAA must delay ripening by competing with the hormone for active sites or by acting as a negative effector on the ripening control mechanism.

Neither kinetin nor GA had any effect on the time of ripening of avocado fruits, as shown in Table 1. They also had no effect on the respiration pattern or ethylene production of fruits except for noticeable stimulation of ethylene production by 10 μM concentration of kinetin. The roles of these two regulators in delaying the ripening of some fruits is well documented (1, 11, 12, 25, 31). Gibberellins have been shown to delay ripening of bananas (25, 31) and tomatoes (1, 11). Because gibberellins and cytokinins have opposing effects (26), this group of hormones has been considered, together with auxins, to control the ripening of fruits (27).

The effects of gibberellins and cytokinins may not delay all aspects of normal ripening but affect only one or a few criteria of ripening. In several cases, only pigment changes of chlorophyll breakdown, or lycopene synthesis have been affected by these hormones (1, 11, 22). Blumenfeld and Gazit (6) and Gazit and Blumenfeld (17) have demonstrated the existence of gibberellins and cytokinins, respectively, in avocado fruits. They have also pointed to the possible synthesis of these hormones in the fruit, particularly in the seed coat. The lack of response of the avocado fruit to exogenous kinetin and GA may be attributable to inadequate endogenous amounts of these hormones. In addition, because cell division continues throughout the life of the avocado fruit, a nonlimiting amount of these hormones, especially cytokinins, in detached fruit is possible.

ABA did not seem to influence the ripening time of intact ‘Hass’ fruits (Table 1) nor respiration pattern or ethylene production of ‘Fuerte’ half fruits (Fig. 3a). This was unexpected because ABA is considered to be a senescence-promoting hormone and was thus assumed to hasten the ripening of the fruits. In tomatoes, ABA accelerated ripening (21, 22) by increasing respiration and ethylene production (21, as well as by stimulating lycopene synthesis (22). Indirect evidence linking ABA to fruit ripening has also been provided by Goldschmidt et al. (18) who reported that an increase in ABA-like inhibitors accompanied senescence of citrus fruits. Information on the role of ABA in ripening of horticultural fruits is still meager; as a result, the extent of its participation in fruit ripening is not yet clear (10).

The results in Table 1 and Figure 3c show that extracts prepared from leaves and peduncle had no effect on the ripening of the fruit. This failure of the extracts to elicit any effect does not necessarily indicate the absence of the ripening inhibitor but rather may be attributable to a weakness of the extraction procedure adopted.

We conclude that the extent of the influence of IAA on avocado ripening by far exceeds that of the other hormones studied. Auxins are likely to be the substances (inhibitors) that prevent ripening of unpicked or freshly harvested avocado fruits. The significance of optimum balance between auxins and the other hormones for the effect to be shown cannot be discounted. The unequivocal identification of this substance must come not only from extraction from the plant but also from confirmation of its effect on unpicked fruits.

Acknowledgment—We thank Drs. J. B. Biale and L. C. Erickson for helpful suggestions and for reading the manuscript. The technical assistance of Cameron Duncan is greatly appreciated.

LITERATURE CITED


