NAVEL ORANGE FRUIT DROP: SECONDARY-FRUIT ONTOGENY, PHYSIOLOGICAL STUDIES, AND GROWTH REGULATOR EFFECTS

By

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To my mother and father, whose encouragement and love made it all possible.

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Chairman: Dr. Frederick S. Davies
Major Department: Horticultural Science (Fruit Crops)

Navel orange (Citrus sinensis (L.) Osbeck) fruit drop, which occurred after the fruit set period, was studied in 2 groves in the north central citrus region of Florida from 1979 to 1982. Fruit drop per year ranged from 63 to 200 fruit per tree. Major causes of drop were secondary-fruit yellowing (SFY), stylar-end decay (SED), and fruit splitting. These causes of fruit drop were related to the unusual structure of the stylar-end of navel orange, which encloses the secondary fruit.

The secondary fruit developed as an extra whorl of carpels within the primary-carpel whorl. A complete secondary pistil was formed entirely within the primary one. Secondary ovary development followed a sigmoid growth curve similar to that of the primary fruit.

An abscission zone was characterized at the base of the secondary fruit, and abscission of the latter occurred prior

to SFY. Secondary-fruit abscission preceded and probably was the cause of primary-fruit abscission in fruit affected by SFY, as indicated by increases in cellulase activity and ethylene levels in the abscission zone of the secondary fruit prior to those in the abscission zone of the primary fruit. Fruit-stem ringing and leaf removal experiments suggested induction of SFY resulted from an interrupted supply of bark-translocated leaf metabolites. Applications of 2,4-dichlorophenoxyacetic acid (2,4-D) to the fruit or fruit-stem reduced induction of SFY from fruit-stem ringing.

Fruit affected by SED and splitting had greater diameter and thicker peel than healthy fruit, and their aperture at the stylar-end had greater diameter. Rind protrusions which usually occurred in segments of fruit affected by SED consisted of secondary-carpel outgrowths and were present early in fruit ontogeny.

A spray application of 20 ppm 2,4-D 5 to 9 weeks after midbloom reduced SFY, but not other causes of fruit drop in the 1980 to 1982 seasons, and increased yield in 1981 with no adverse effect on fruit quality. Other 2,4-D, gibberellic acid (GA), or 2,4-D + GA sprays at or within 19 weeks of midbloom were less effective or ineffective.

INTRODUCTION

Navels constitute a group of early- to midseason-maturing sweet orange cultivars (Citrus sinensis (L.) Osbeck) characterized by the presence of a small, secondary fruit, the navel, at the stylar end of the primary or main fruit. Navel oranges are commercially grown throughout most of the citrus areas of the world (51). Yields, however, are usually erratic and lower than for other sweet oranges (23, 30,100,119). Navel yields in Florida are typically greater than those in other citrus areas but frequently can be low and fall below those of 'Hamlin' or 'Valencia' (67,68). Although most authors attribute low navel yields to poor fruit set (23,30,67,73,119), navel orange has been shown to have significant fruit drop after the fruit-set period (79).

Causes of navel fruit drop after the fruit set period in Florida include secondary-fruit yellowing (SFY), stylar-end decay and fruit splitting (77,78,79). These fruit drop problems are related to the unique structure of the stylar end of the navel fruit, which encloses the secondary fruit (77,79). Bouma (14) and Holtzhausen (52) studied fruit

development of navel orange, but little has been done regarding secondary-fruit ontogeny (118).

Secondary-fruit yellowing accounted for more than half of the fruit drop after fruit set in a navel grove in Florida, in 1979 (77). The problem was related to physical separation of the secondary from the primary fruit (77,79). Pest or disease factors did not seem to be involved (77, 112). Fruit affected by SFY produced high amounts of ethylene (112), but other physiological aspects of SFY and related secondary-fruit separation have not been studied.

The objectives of this research were to study secondary-fruit ontogeny, to investigate some physiological aspects of SFY, and to control navel orange fruit drop in Florida using growth regulator applications.

CHAPTER I

SECONDARY-FRUIT ONTOGENY

Introduction

Navel orange fruit drop after the fruit-set period has been related to the unique structure of the fruit stylar-end and of the secondary fruit (77,78,79). Secondary-fruit yellowing, an important cause of fruit drop during late spring and early summer, may result from the physiological separation of the secondary fruit from the primary fruit. This separation possibly involves an abscission zone at the base of the secondary fruit (77,79). Fruit with larger stylar-end aperture are more affected by stylar-end decay and fruit splitting, which are important causes of navel fruit drop during late summer and early fall (79). In addition, rind-like tissue protrusions that originate from the secondary fruit occur in the decayed primary-fruit locules of most fruit affected by stylar-end decay (77,79).

Bouma (14) in Australia, and Holtzhausen (52) in South Africa, studied fruit development of navel orange but made

no reference to the secondary fruit. Some aspects of secondary-fruit structure and development have been briefly discussed (12,105,118), but the available information on the ontogeny of the secondary fruit is limited.

Developmental morphology of the secondary fruit in navel orange was studied with emphasis on early ontogeny, characterization of a secondary-fruit abscission zone and the occurrence of secondary-fruit tissue protrusions within the primary-fruit locules.

Literature Review

Fruit Ontogeny

Flower induction in sweet oranges typically occurs during late fall and early winter (8). Differentiation of the vegetative meristem into a reproductive meristem, however, occurs only at the onset of the spring growth flush (4). The floral apical meristem is characterized by a flattening of the apical dome and the production of closely-spaced successive whorls of flower organ primordia. Each whorl of primordia is located slightly above and inside the preceding whorl and gives rise to the various floral organs (105).

The innermost whorl of primordia of floral organs around the floral meristem consists of ca. 10 carpel

primordia (105). These primordia are first observed around the floral meristem when the flower bud is 1-1.5 mm in diameter. The carpel primordia are initially free, finger-like and inwardly curved. Marginal meristems on the sides of each primordium produce lamina-like wings which grow toward the expanding apical meristem or axis (105). Developing carpels appear horseshoe-shaped in transection with the open ends toward the center of the meristem. The carpel surface at the periphery of the meristem is analogous to the abaxial leaf surface (34). Carpels become fused at the abaxial surface of their lamina-like wings, and are fused to the expanding floral axis (i.e., the central axis) by their margins (37). Carpels develop as chambers that are open at the top, and are fused into one single pistil, which composes the gynoecium of the flower.

A fully developed citrus pistil has a well-differentiated ovary, style and stigma. Each carpellary chamber is a locule in the ovary, and the fused wings of the carpels become the septa (105). The carpellary chambers are continuous throughout the style and stigma and form the stylar canals. Stylar canals, one per carpel, open onto the surface of the stigma and on the upper portion of the locules between the two rows of ovules (37).

Ovules develop early in the ontogeny of the carpels from tissue that is produced by the fusion of the 2 carpellary margins to the central axis in the ovary (105).

Two rows of ovules are present per locule. Placentation is axile.

Juice sacs develop from the adaxial carpel surface as club-shaped emergences prior to or at anthesis. They occupy the entire locule when the ovaries are about 10 mm in diameter (37).

A secondary set of carpels develops in some instances at the apex of the floral axis within the primary pistil (118). These secondary carpels become the characteristic navel or secondary fruit of some citrus cultivars, such as in navel orange (51).

Fruit Morphology and Anatomy

A citrus fruit is a hesperidium,which differs from other berries by the presence of a hard or leathery rind (105). The pericarp is composed of the exocarp, mesocarp and endocarp. The exocarp, or flavedo, is the pigmented outer portion of the fruit derived from the abaxial surface of the carpels and is composed of an epidermis and several layers of cells adjacent to it. The epidermis is covered by a thick waxy cuticle and contains stomata interspersed among epidermal cells (106). Some authors (12,106) recognize a hypodermis composed of 1 to 3 rows of collenchyma cells. Several layers of chlorenchyma cells, oil glands and the endings of vascular bundles occur beneath the hypodermis.

Chlorophyll and carotenoid pigments in the chlorenchyma cells give the fruit its characteristic green to orange color (32).

The mesocarp, or albedo, is the usually white, spongy tissue found internal to the exocarp. This tissue is analogous to the spongy mesophyll of a leaf. The mesocarp is composed of aerenchyma tissue which consists of lobed parenchyma cells and schizogenous intercellular spaces (105). An extensive network of vascular bundles permeatesthe albedo (12). The exocarp plus the mesocarp are commonly referred to as the peel or rind of a citrus fruit.

The endocarp originates from the adaxial surface of the carpel primordia and surrounds the locular cavities. This tissue is composed of an inner epidermis and several layers of parenchyma cells (105). A portion of the endocarp differentiates into the membrane that lines the locules. Juice sacs develop in the endocarp (primarily on the dorsal wall of the locules) from the epidermis and adjoining cell layers. The locules of a fruit are separated by septa formed by fusion of adjacent carpellary walls. A locule that contains juice sacs, seeds, and the surrounding membrane is called a segment (105). Segments are clustered around the central axis of the fruit and form the edible pulp of the latter. The central axis originates from expansion of the floral meristem. Both the central axis and the portion of the septa between locular membranes are composed of mesocarp-like aerenchyma tissue (12).

Major vascular bundles in a citrus fruit are restricted to the mesocarp and the central axis. Five bundles occur in each carpel (37). Carpellary bundles in the mesocarp consist of a prominent dorsal bundle opposite the locule and two septal or lateral bundles opposite the septa. Two marginal bundles opposite the septa or carpel margins occur in the distal half of the central axis. Lateral bundles of 2 adjacent carpels, as well as two adjacent marginal bundles, are fused. All major carpellary bundles are collateral bundles (37). Dorsal and lateral bundles diverge from axial bundles at the base of the ovary (37). Axial bundles, however, continue into the proximal half of the central axis of the fruit and then terminate after giving rise to ovular traces and marginal bundles (105). The marginal bundles are inverted collateral bundles which branch and fuse with septal bundles and then give rise to concentric stylar bundles (37).

Fruit Growth and Development

The growth of a citrus fruit, as determined by changes in volume, equatorial diameter or dry or fresh weight, typically follows a sigmoid pattern (10,14,52). Bouma (14) and Holtzhausen (52) defined three stages of development for navel orange. Stage I, the cell division stage, lasts until a few weeks after anthesis and is characterized by small

growth rates. Fruit growth during stage I is due mainly to growth of the peel that results from cell division and enlargement in the mesocarp and exocarp tissues. The majority of cells found in a fully developed fruit is produced during stage I, although cell divisions continue in the outer peel throughout fruit development. Stage II is characterized primarily by rapid enlargement of cells in the fruit segments and lasts until color break. At the end of this stage of development the fruit has almost attained final diameter and dry weight. The peel reaches maximum thickness early in stage II and then decreases to almost final thickness. Stage III is characterized by slow fruit growth, and the latter is due primarily to development of the fruit segments. The rind becomes orange during stage III, accompanied by a decrease in titratable acidity of the juice and other changes indicative of maturity. The fruit typically reach maximum fresh weight during stage III, and then fresh weight gradually decreases.

Fruit Abscission

Detachment of the young citrus fruit from the plant usually is caused by the separation of cells in one of the 2 abscission zones which are located at the base of the pedicel and at the base of the ovary (32). Sclerenchyma tissue which forms in the abscission zone at the base of

the pedicel limits abscission to the abscission zone at the base of the developing fruit (19).

The abscission or separation zone is the zone of tissues proximal to the structure to be shed. This zone may or may not be anatomically distinguishable from adjacent tissues prior to abscission (11,123). The abscission zone is composed typically of an epidermis, cortex, vascular bundles and pith (11,34,72). The epidermis and pith are usually similar to corresponding tissues on either side of the abscission zone. Cortical parenchyma cells in the abscission zone, however, usually are close-packed, thin-walled, densely protoplasmic, and smaller than adjacent cells. Xylary and phloic fibers of vascular bundles in the abscission zone are unusually small or absent, and the number of vessels reduced (17,80). The separation or abscission layer typically refers to 1 or 2 layers of cells in which separation actually occurs. The protective layer is the layer of suberized cells which develop over exposed surfaces of the plant following fruit abscission. The protective layer typically consists of a periderm which is usually continuous with the periderm of the stem (11,34).

The abscission zone of a citrus fruit is not easily distinguished from adjacent tissues by anatomical characteristics (124). Parenchyma cells in the separation zone often are slightly smaller than in surrounding tissue, and the tracheary elements in the abscission zone are compressed. The occurrence of starch grains in parenchyma

cells of the separation layer is the first obvious indication of an abscission zone in citrus (19,124). Starch grains can be observed in cortical parenchyma cells on both sides of the separation layer and in the intercellular spaces between separated cells after abscission has occurred. No cell division has been detected in association with citrus fruit abscission (19,124).

No protective layer forms in the proximal (stem) side after fruit abscission in citrus, and the pedicel usually dessicates and dies (124). A protective layer does develop, however, on the distal (fruit) side of the abscission zone (19,124).

Materials and Methods

Navel orange trees (Citrus sinensis (L.) Osbeck) 12- to 25-years-old, on sour orange rootstock (Citrus aurantium L.) were used in this study. The trees were growing in a grove near Eustis (Lake County), in the north central citrus region of Florida.

Samples for secondary-fruit ontogenetic studies were collected from late February until fruit maturity in late October, from 1980 to 1982. Two to 10 flower buds or fruit per tree from 5 to 30 randomly selected trees were collected daily until anthesis, weekly until stylar abscission and then monthly.

Anatomical studies of secondary-fruit separation from the primary-fruit were done on samples of tissues from the central axis of the primary fruit, taken just below the secondary ovary. Approximately 50 fruit which were sampled at different stages of secondary-fruit yellowing during June and July were examined in 1981 and 1982.

Primary- and secondary-fruit development were followed by measuring their equatorial diameter on 30 randomly sampled fruit weekly. Stylar-end aperture diameter was also measured on these fruit.

Light Microscopy

Plant material for anatomical and ontogenetic studies was fixed in formalin-acetic acid-50% ethanol (5:5:90 v:v:v), for a few days to several months. The material was rinsed in 50% ethanol, dehydrated through an ethanol series, infiltrated with paraffin, sectioned on a rotary microtome at 8 to 25 µm, mounted on glass slides and stained with either safranin-fast green (58,102), ruthenium red (57), or toluidine blue (91). In addition, iodine-potassium iodide (IKI) staining (57) was utilized in abscission zone studies. Hesperidin crystals, which form during tissue dehydration and interfere with the study of thin sections (37), were removed by adding 0.25% NaOH to a 50% ethanol step in the staining sequence (104).

Scanning Electron Microscopy

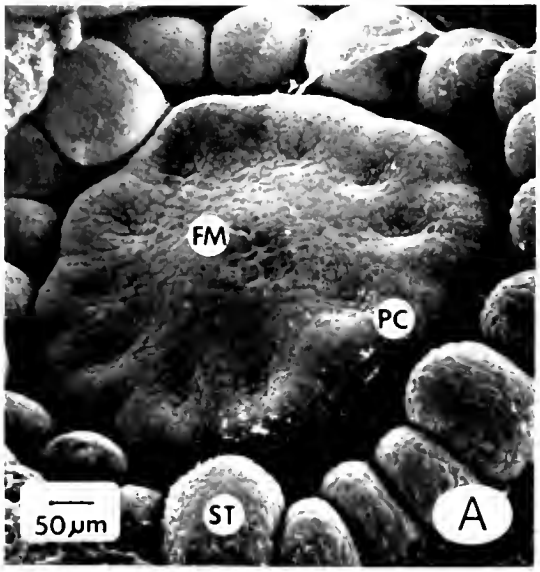
Flower buds and young pistils for studies at the scanning electron microscope were killed and fixed in 2% glutaraldehyde in 0.1 M cacodylate buffer (pH 7.2) at 20 to 25 C for 24 hrs, rinsed with buffer for 30 min, post-fixed in 1% osmium tetroxide for 2 to 3 days, dehydrated through an ethanol series, critical-point dried, and gold coated. Specimens were examined in a Hitachi S-450 scanning electron microscope operated at 20 KV.

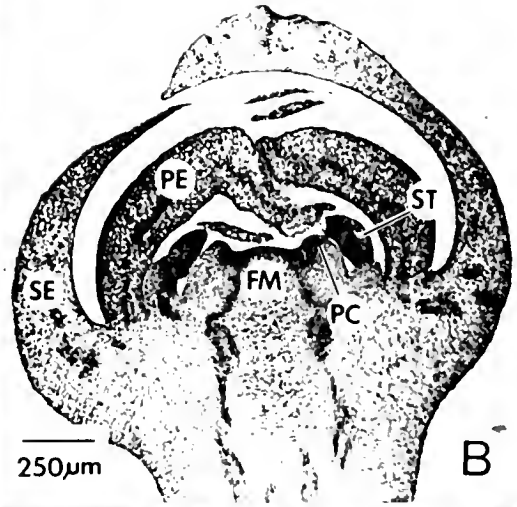
Results and Discussion

Secondary-Fruit Ontogeny

Primary-carpel primordia of navel orange were first observed as the innermost whorl of primordia around the floral apical meristem in flower buds ca. 0.8 mm in length (Fig. 1.1 A). Carpel primordia in longisections of 1 mm-length flower buds were finger-like protuberances around the floral meristem and inwardly curved (Fig. 1.1 B). Marginal meristems on the primordia produced lamina-like wings which grew toward the center of the apical meristem (Fig. 1.1 C). Carpels were initially free, but the wings of adjacent

Fig. 1.1. Primary-carpel primordia in flower buds 24 to 26 days before anthesis in navel orange. A, 0.8 mm-length flower bud; B-D, 1 mm-length flower bud: B, longisection, C, top view, and D, top view; E-F, 1.2 mm-length flower bud: E, transection, and F, top view. FM, floral apical meristem; PE, petal; PC, primary-carpel primordium; SE, sepal; ST, stamen primordium.


FM
PC
ST
50µm
A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SE
FM
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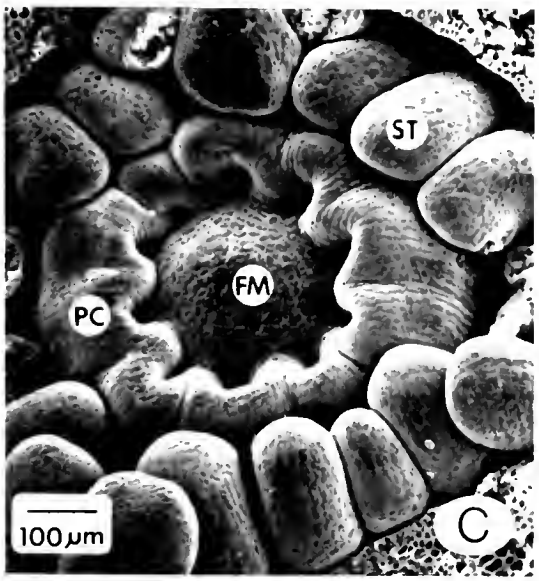
B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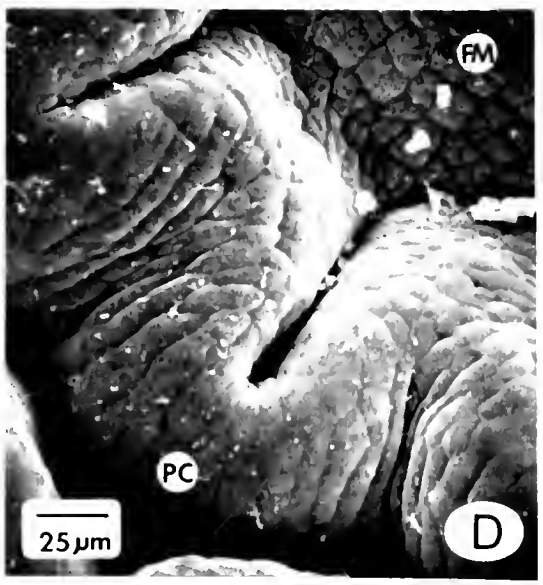
C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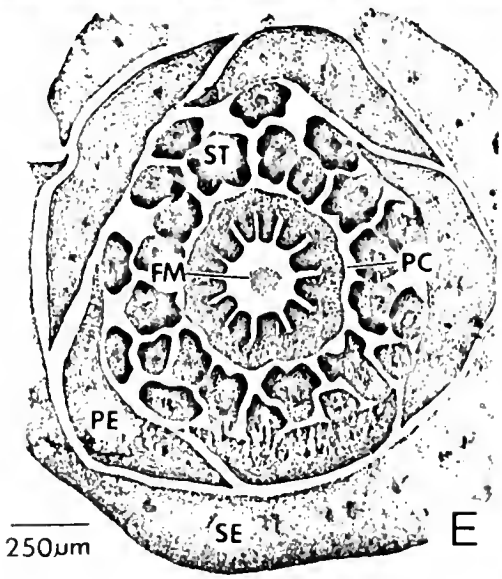
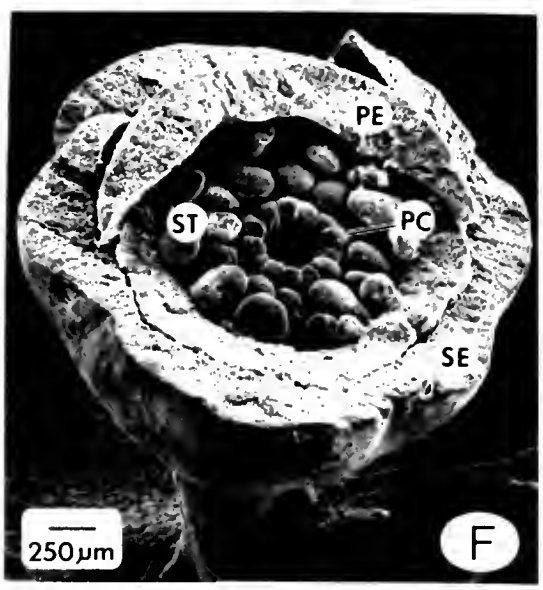
PC
25µm
D


ST
FM
PC
PE
SE
250µm
E


PE
ST
PC
SE
250µm
F

carpels became fused early in their ontogeny (Fig. 1.1 D). Carpel primordia were horseshoe-shaped in transection with the open ends toward the center of the floral meristem (Fig. 1.1 E,F).

Secondary-carpel primordia were first observed within the primary-carpel whorl in flower buds approximately 1.5 mm in length (Fig. 1.2 A,B,C). No sepal, petal or stamen primordia were associated with secondary-carpel development. Secondary-carpel primordia in longisections of flower buds 1.8 to 2 mm in length (Fig. 1.2 D,E,F) were finger-like, inwardly curved protuberances around the floral apical meristem. Marginal meristems on the primordia produced lamina-like wings which grew toward the center of the floral meristem (Fig. 1.2 F) in a manner similar to primary-carpel primordia (37,105). Secondary-carpel primordia in transections of developing pistils from 2.7 to 3 mm-length flower buds (Fig. 1.3 A,B,C,D) appeared horseshoe-shaped with the open ends toward the center of the meristem (Fig. 1.3 B,E). Secondary carpels were initially free (Fig. 1.3 B, E,F), but their lamina-like wings fused with one another early in their ontogeny.

Secondary carpels were entirely within primary pistils in flower buds 6 to 8 mm in length (Fig. 1.4 A,B,C,D,E,F). Contrary to the uniform and symmetrical development of primary carpels (37,105), secondary carpels grew asymmetrically and frequently overlapped each other (Fig. 1.4 D,E,F). The space for secondary-carpel development was

Fig. 1.2. Secondary-carpel ontogeny in flower buds 21 to 23 days before anthesis in navel orange. A-C, 1.5 mm-length flower buds: A, flower bud, B, secondary-carpel primordia, and C, longisection; D-E, 1.8 mm-length flower bud: D, secondary-carpel primordia, and E, longisection; F, 2 mm-length flower bud, secondary-carpel primordia. FM, floral apical meristem; PC, primary-carpel primordium; PE, petal; SC, secondary-carpel primordium; ST, stamen.

A
250 µm
B
SC
FM
PC
50 µm
C
PE
SC
PC
ST
FM
250 µm
D
PC
SC
FM
25 µm
E
PE
PC
ST
SC
FM
250 µm
F
SC
FM
25 µm

Fig. 1.3. Secondary-carpel ontogeny in flower buds 18 to 19 days before anthesis in navel orange. A-B, 2.7 mm-length flower buds: A, primary pistil, and B, secondary-carpel primordia; C-F, 3mm-length flower buds: C, primary pistil, D, longisection of primary pistil, E, secondary-carpel primordia, and F, secondary-carpel primordia. FM, floral apical meristem; PC, primary carpel; PP, primary pistil; SC, secondary-carpel primordium.

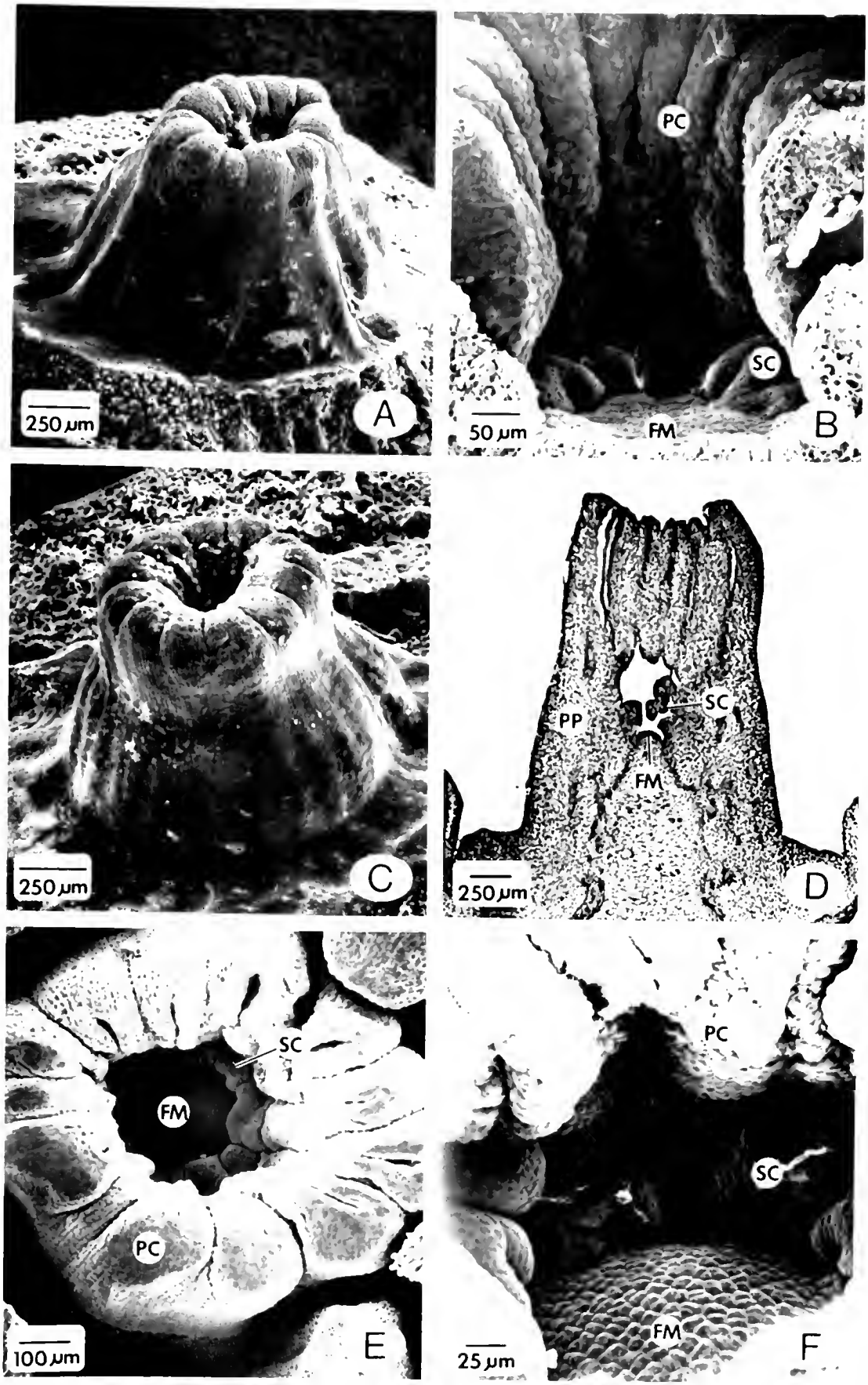
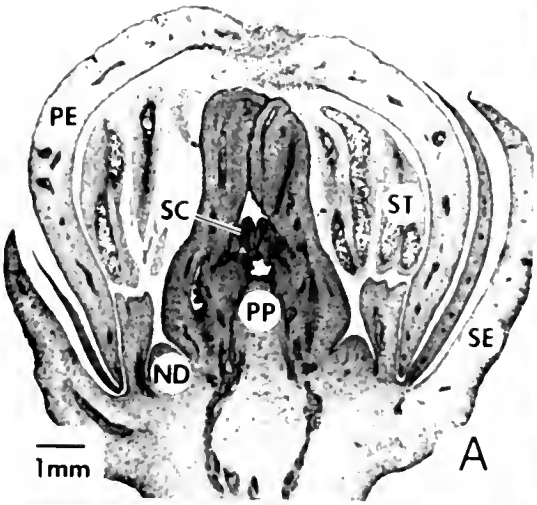
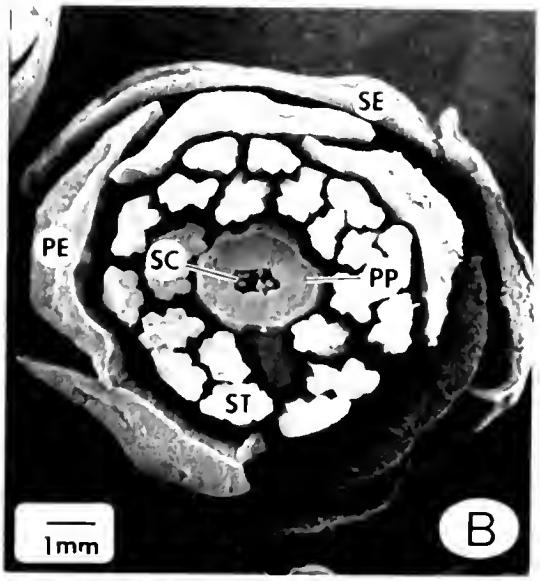
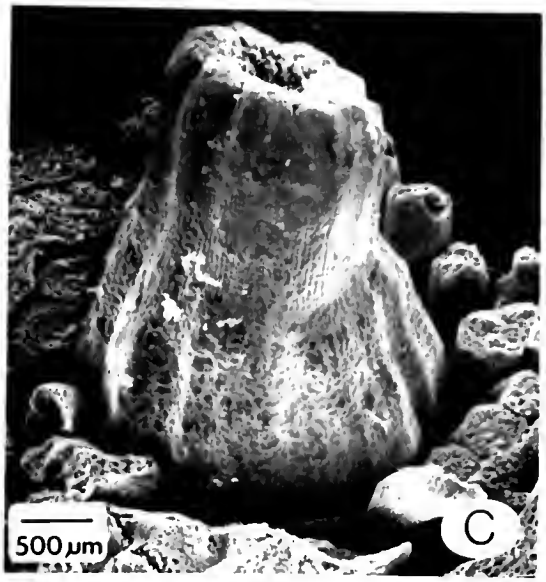
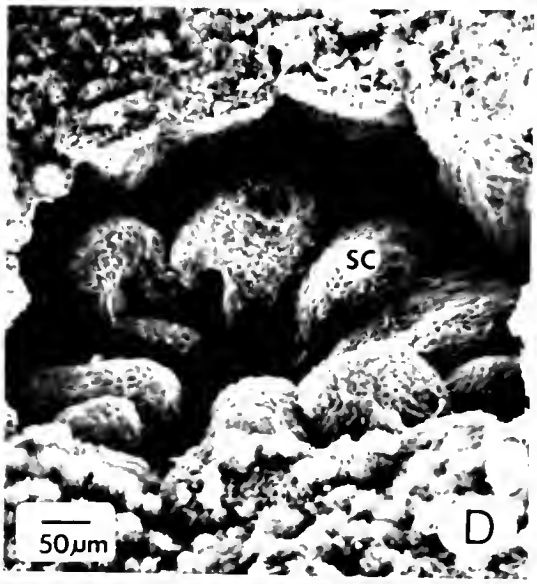

250 µm
A
PC
SC
50 µm
FM
B
250 µm
C
PP
SC
FM
250 µm
D
SC
FM
PC
100 µm
E
PC
SC
FM
25 µm
F

Fig. 1.4. Secondary-carpel ontogeny in flower buds 12 to 15 days before anthesis in navel orange. A-D, 6 mm-length flower bud: A, longisection, B, transection, C, primary pistil, and D, secondary carpels; E-F, 8 mm-length flower buds: E, longisection of primary pistil, and F, secondary carpels. ND, nectar disc; PE, petal; PO, primary ovary; PP, primary pistil; SC, secondary carpel; SE, sepal; ST, stamen.


PE
SC
ST
PP
SE
ND
1mm
A


SE
PE
SC
PP
ST
1mm
B


500µm
C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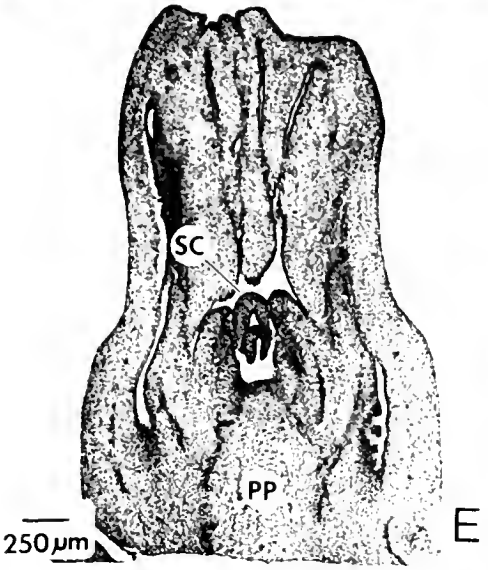
SC
PP
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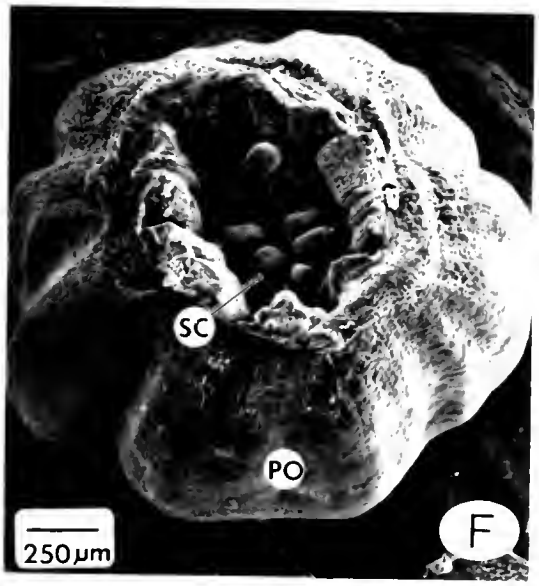
SC
PO
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F

limited and, in some instances, a carpel bent over and grew toward the floral meristem (Fig. 1.5 A). Most secondary carpels remained partially or totally free above the base (Fig. 1.5 B,C,D), although fusion at their base (secondary-ovary region) was generally complete (Fig. 1.5 B,D).

Secondary carpels generally developed into a complete secondary pistil entirely within the primary pistil before anthesis (Fig. 1.6 A). Incomplete fusion of secondary carpels frequently resulted in multiple secondary styles and stigmas (Fig. 1.6 A,B,C,D) and a single, well-fused secondary ovary (Fig. 1.6 E,F). Conversely, primary carpels are almost always fused and develop into pistils with a single ovary, style, and stigma (37,105).

Stylar canals were present in the stigmas and styles of secondary pistils (Fig. 1.6 B). As in primary carpels (37, 105), the stylar canals of secondary styles opened into the locules, and formed continuous chambers within secondary carpels. Stigmatic surfaces composed of papillose hairs were present on the distal half of the secondary styles (Fig. 1.6 C,D). Secondary styles and stigmas were less distinct than the primary ones (Fig. 1.7 A,B,C). The constriction at the distal end of the ovary that marks the beginning of the style was less noticeable (Fig. 1.7 A,C,D), and generally there was no expansion of the distal extremity of the styles to delineate the stigma (Fig. 1.7 B,C).

Expansion of the secondary style caused at times splitting of the primary style. Abscission of the primary

Fig. 1.5. Secondary-pistil ontogeny in flower buds 5 to 6 days before anthesis in navel orange. A, 10 mm-length flower bud, longisection of primary pistil and secondary carpels; B-D, 12 mm-length flower bud: B, longisection of primary and secondary pistils, C, transection of primary and secondary ovaries, and D, longisection of secondary pistil. AX, axial vascular bundle; FM, floral apical meristem; ND, nectar disc; PG, primary stigma; PO, primary ovary; PS, primary style; SC, secondary carpel; ST, stamen; TC, tertiary carpel primordium.

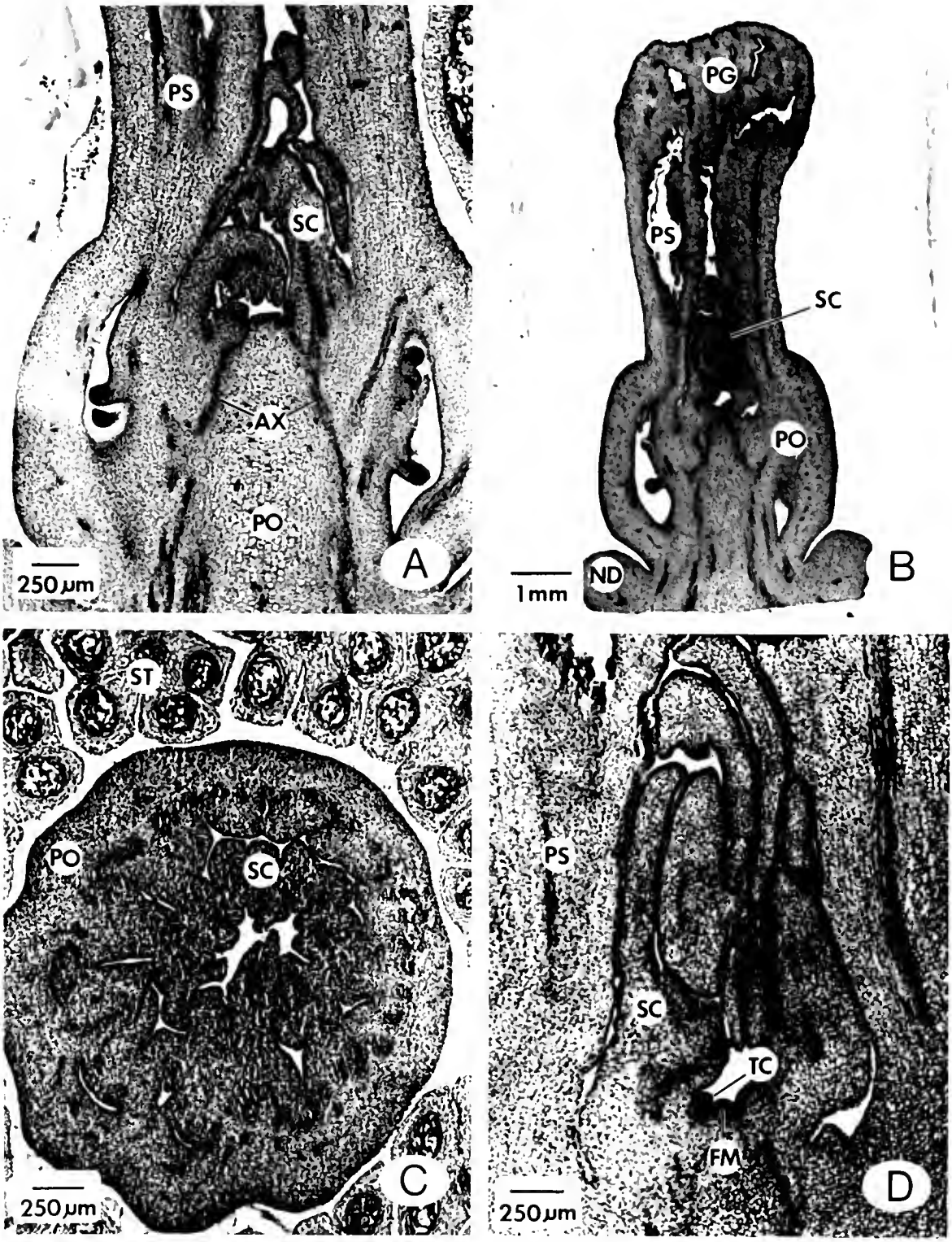

PS
SC
AX
PO
250 µm
A
PG
PS
SC
PO
ND
1mm
B
ST
PO
SC
250 µm
C
PS
SC
TC
FM
250 µm
D

Fig. 1.6. Secondary-pistil ontogeny in flower buds 4 days before anthesis in navel orange. A-F, 15 mm-length flower buds: A, longisection of primary pistil, B, transection of primary and secondary styles, C, longisection of primary style and secondary stigmas, D, transection of primary style and secondary stigmas, E, transection of flower bud, and F, longisection of secondary ovary. AX, axial vascular bundle; CA, stylar canal; DB, dorsal vascular bundle; PE, petal; PO, primary ovary; PP, primary pistil; PS, primary style; SE, sepal; SG, secondary stigma; SL, secondary-ovary locule; SO, secondary ovary; SP, secondary pistil; SS, secondary style; ST, stamen.

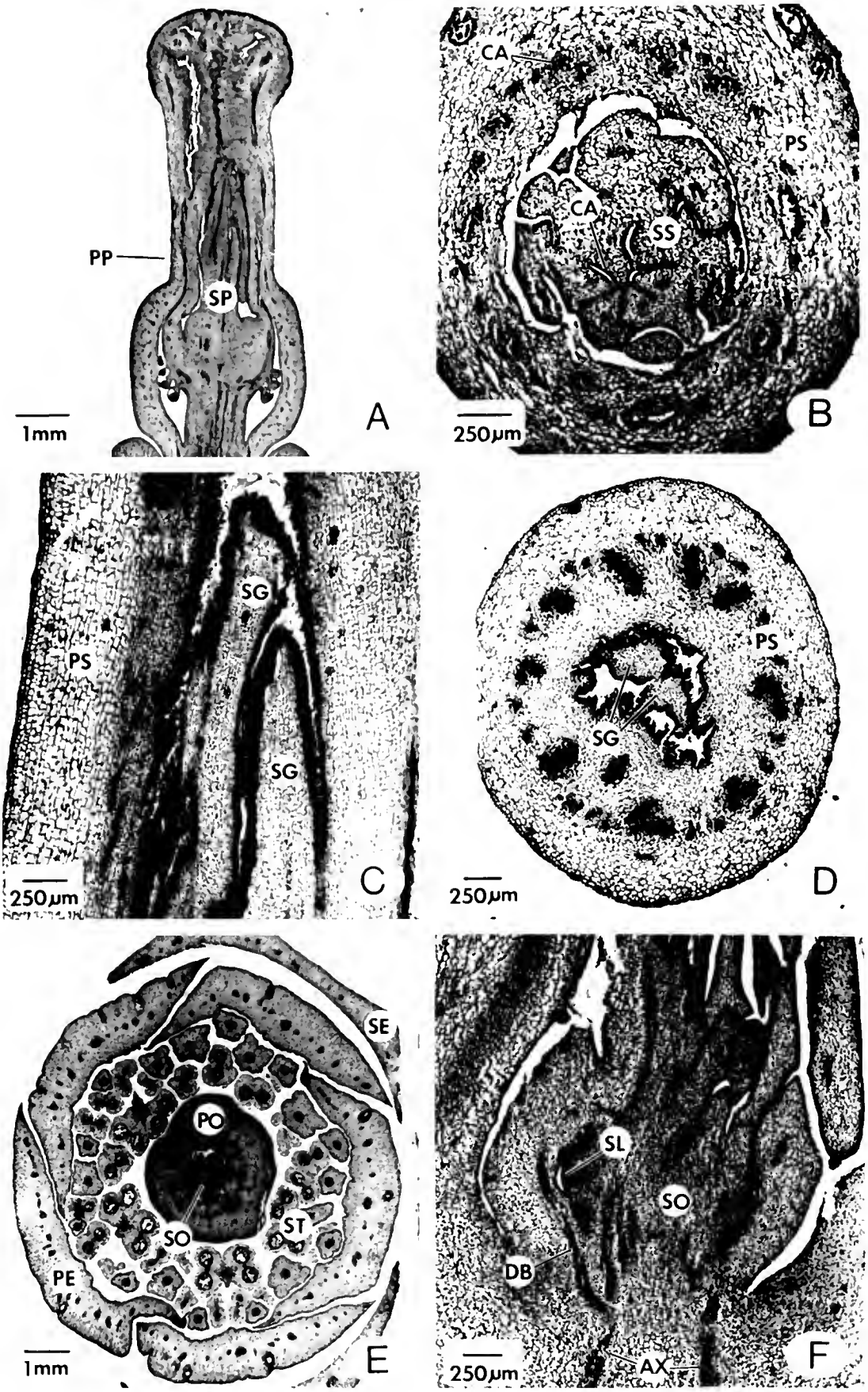
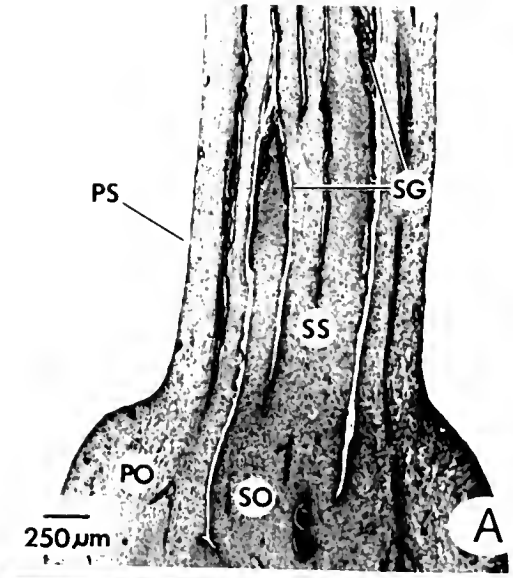
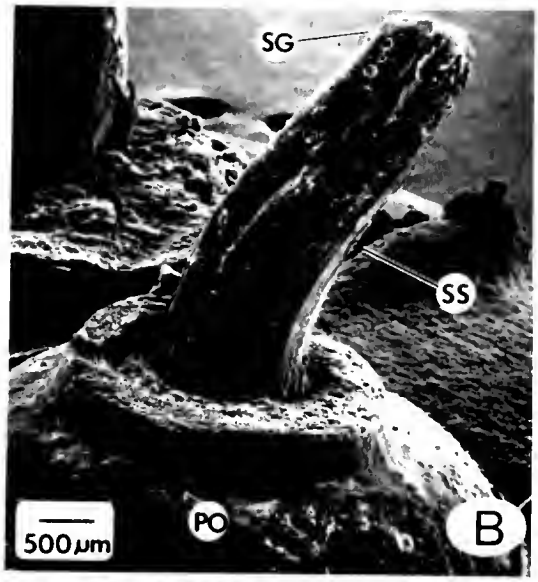
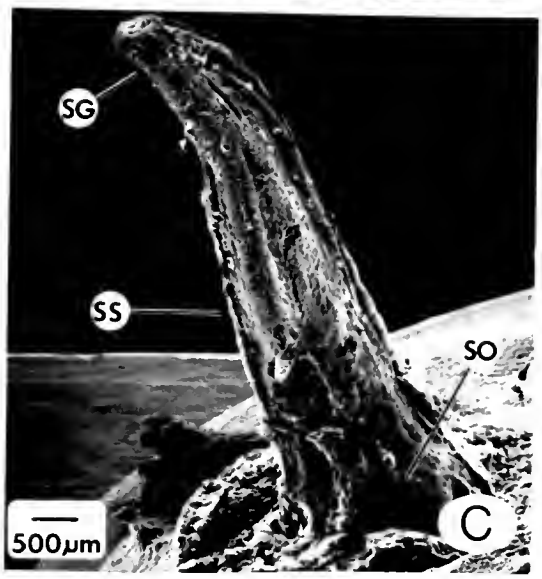
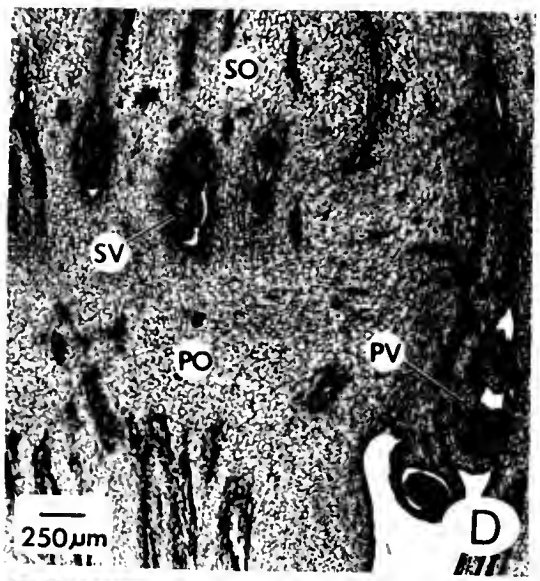

PP
SP
1mm
A
CA
PS
CA
SS
250µm
B
SG
PS
SG
250µm
C
PS
SG
250µm
D
SE
PO
SO
ST
PE
1mm
E
SL
SO
DB
AX
250µm
F

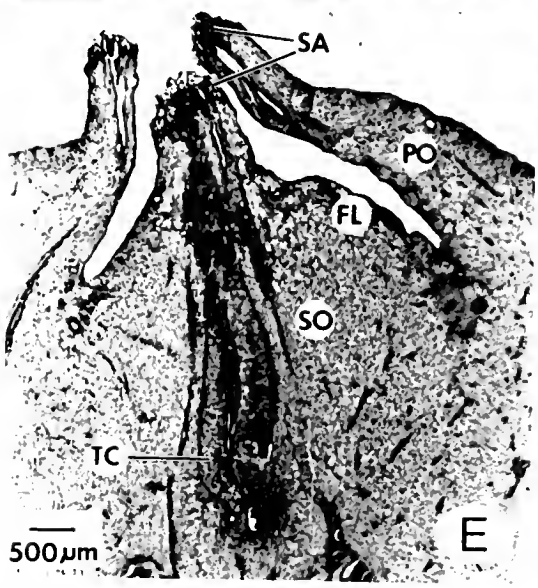
Fig. 1.7. Secondary-pistil development in flower buds 3 days before anthesis to 10 days after petal fall in navel orange. A-B, 16 mm-length flower buds, 3 days before anthesis: A, longisection of primary and secondary pistil, and B, secondary style and stigma; C-D, at anthesis: C, secondary pistil, and D, longisection of primary and secondary ovaries; E-F, at 10 days after petal fall: E, longisection of primary and secondary ovaries, and F, longisection at the stylar-end of primary and secondary ovaries. FL, flavedo; PO, primary ovary; PS, primary style; PV, primary ovule; SA, stylar abscission zone; SG, secondary stigma; SO, secondary ovary; SS, secondary style; SV, secondary ovule; TC, tertiary carpel; TG, tertiary stigma.

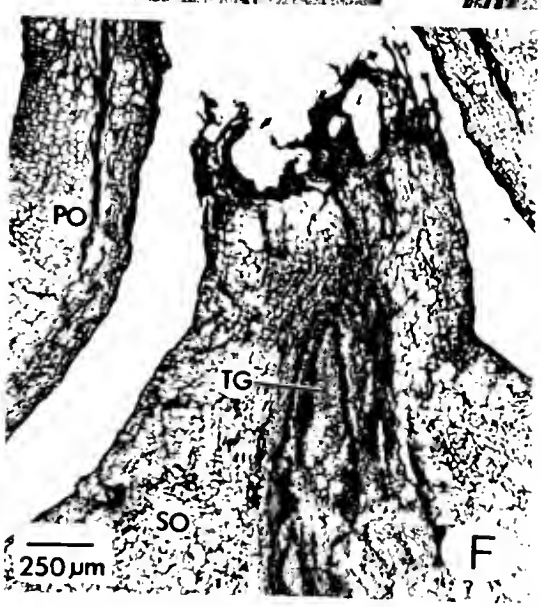

PS
SG
SS
PO
SO
250 µm
A


SG
SS
PO
500 µm
B


SG
SS
SO
500 µm
C


SO
SV
PO
PV
250 µm
D


SA
PO
FL
SO
TC
500 µm
E

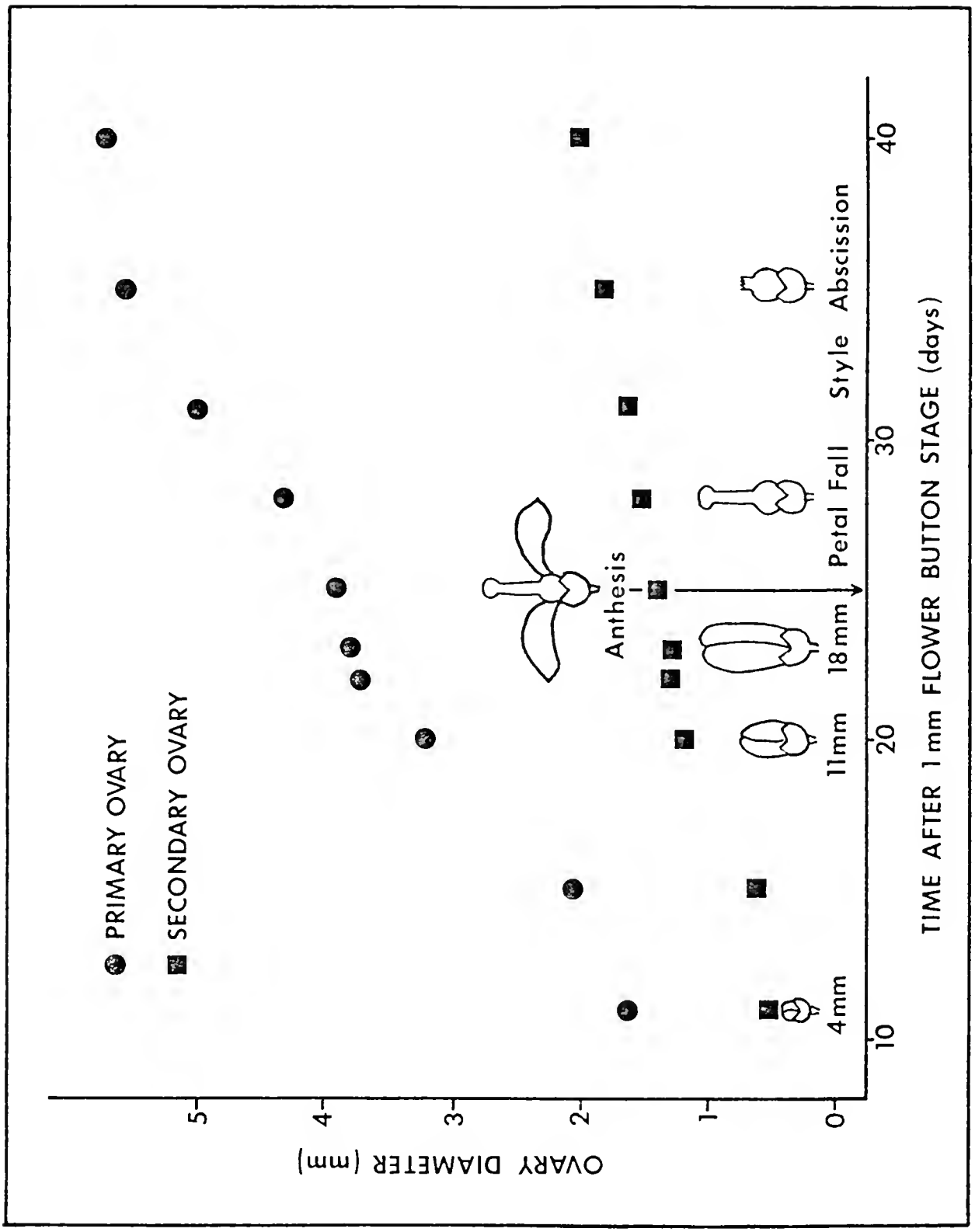

PO
TG
SO
250 µm
F

style occurred about 8 days after petal fall and usually preceded secondary-style abscission (Fig. 1.7 E,F). Secondary styles, however, were simply broken off by the detachment of the primary style in some instances.

Secondary ovaries were generally well differentiated at the base of secondary pistils (Fig. 1.7 C,D). These ovaries were ca. 1.5 mm in diameter at anthesis (Fig. 8).

Pericarp tissues of the mature secondary ovary were similar anatomically to primary-ovary tissues. Most secondary fruit had their mesocarp partially fused to primary-fruit mesocarp at the stylar end of the primary fruit (Fig. 1.7 D,E). Flavedo or exocarp tissue developed on the abaxial surface of the secondary ovary in areas where no fusion to the primary ovary occurred (Fig. 1.7 E). The distal half of a secondary ovary typically had a well-developed exocarp (Fig. 1.7 E,F). Secondary-fruit endocarp consisted of segments clustered around a central axis. As in the primary fruit (105), segments were composed of a membrane-lined locule, which contained juice sacs and seeds, and were separated by septa. Juice sacs were observed shortly after anthesis as randomly arranged, dome-shaped protuberances from the locular wall which is adjacent to the mesocarp. The juice sacs in fully developed secondary fruit, however, were arranged in clusters along the major vascular bundles which abut the wall of the segment.

Fig. 1.8. Primary- and secondary-ovary diameter after the 1 mm flower bud stage in navel orange. Primary- and secondary-carpel primordia are first observed in the 1 mm- and 2 mm-length flower bud stages, respectively. Flower-bud and primary-pistil stages are indicated at the bottom. Each value represents the mean of measurements in 12-20 ovaries.


PRIMARY OVARY
SECONDARY OVARY
OVARY DIAMETER (mm)
0
1
2
3
4
5
10
20
30
40
TIME AFTER 1 mm FLOWER BUTTON STAGE (days)
4mm
11mm
18mm
Anthesis
Petal Fall
Style Abscission

The secondary-fruit central axis was an extension of the primary-fruit one. In fact, the latter axis functions as the pedicel for the secondary fruit (23).

Axial bundles of the primary fruit continued acropetally into the distal half of the central axis of the primary fruit and entered the secondary ovary (Fig. 1.6 F). These bundles, however, typically terminate after giving rise to ovular traces and marginal bundles in primary fruit without a secondary fruit (37,105). Each secondary carpel had 5 major vascular bundles. Bundles in the mesocarp consisted of a dorsal bundle opposite the locule and 2 lateral bundles opposite the septa. Two marginal bundles opposite the septa or the carpellary margins occurred in the distal half of the central axis of the secondary fruit. All major carpellary bundles were collateral bundles, and marginal bundles were inverted. Dorsal and lateral bundles diverged from axial bundles at the base of the secondary ovary. Marginal bundles, however, diverged from axial bundles after the latter had penetrated the proximal half of the central axis of the secondary fruit. This type of carpellary vascularization is similar to that of the primary carpels as described by Schneider (105). As in the primary fruit, lateral bundles of 2 adjacent carpels, as well as 2 adjacent marginal bundles, were fused. In addition to the major vascular bundles, minor ones extended from the primary fruit into the secondary fruit through their fused mesocarps.

Considerable variability was observed in the size and development of the secondary fruit. Secondary fruit ranged from barely noticeable rind tissue only to well-developed fruit similar to the primary fruit, but much smaller.

Development of the secondary ovary into the secondary fruit results in a unique fruit structure in navel orange. The secondary fruit typically is contained within the stylar end of the primary fruit. As a result, this stylar end usually is open. The borders of the stylar-end aperture and the tissues lining the stylar-end cavity are extensions of the rind of the primary and secondary fruit. The aperture at the stylar-end of the primary fruit varied from very small or absent, to about 50 mm in diameter. The peel at the stylar-end of the primary fruit extended over the secondary fruit in some instances. As a result, small apertures sometimes enclosed large secondary fruit. Conversely, large stylar-end apertures sometimes exposed small secondary fruit. No correlation has been found between the size of the stylar-end aperture and that of the secondary fruit (77).

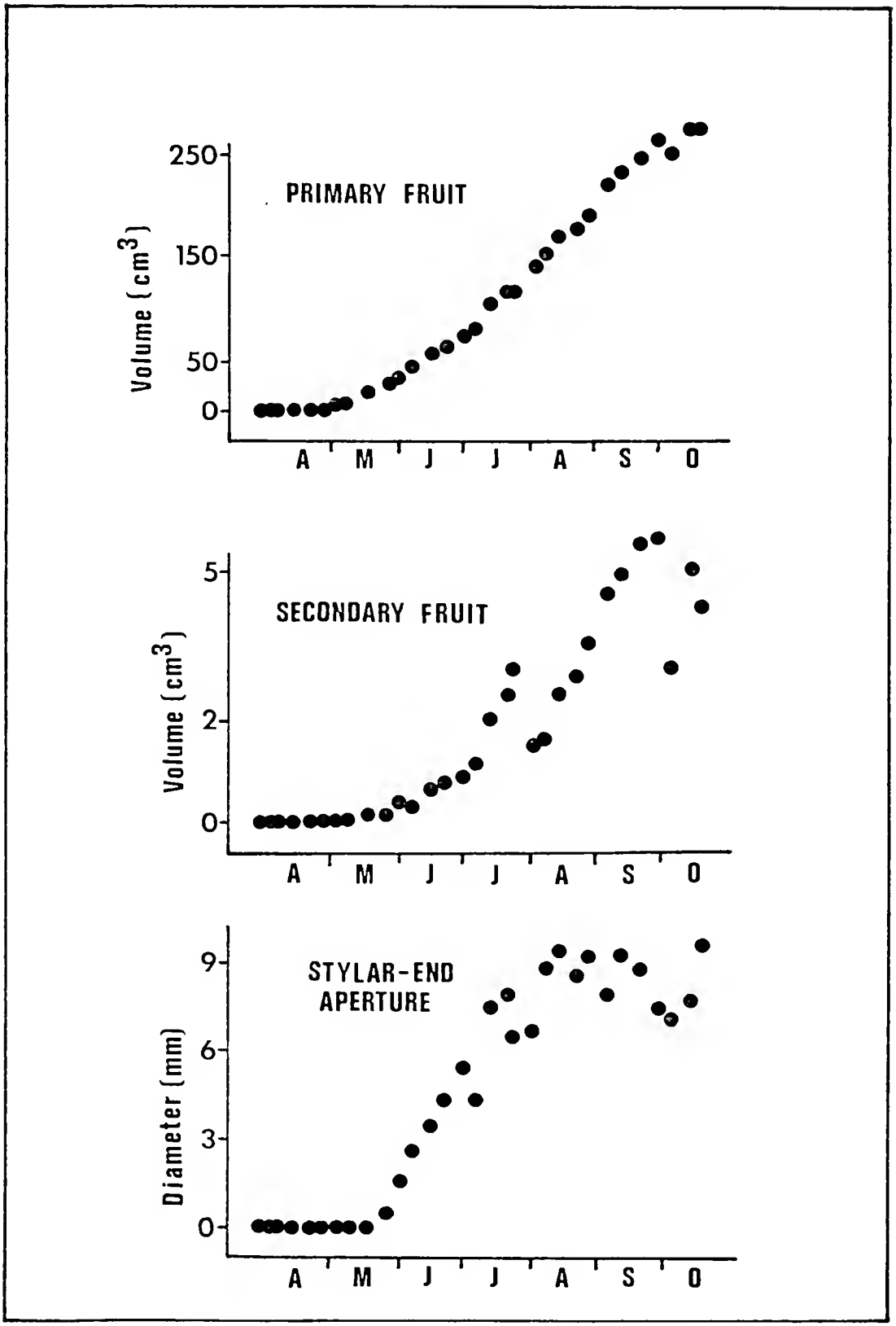
The stylar-end of secondary fruit varied from ridged to hemispherical and rarely exhibited a stylar-end aperture. A ridged stylar end usually reflected incomplete fusion of secondary carpels. Each ridge represented the distal portion of a secondary carpel. Similarly, carpels of the primary and secondary fruit of the 'Buddha's Hand' citron are partially free, and their distal portions vary from ridged to finger-like (51).

A tertiary fruit frequently was observed within well-developed secondary fruit. Tertiary carpel primordia were observed as an additional whorl of primordia within the secondary-carpel whorl around the floral meristem (Fig. 1.5 D). Tertiary carpels developed into complete pistils entirely within secondary pistils shortly after anthesis (Fig. 1.7 E,F). The ontogeny of the tertiary fruit was similar to that of the secondary fruit.

Secondary-Fruit Development

Primary and secondary fruit in navel orange followed a sigmoid pattern of growth (Fig. 1.9) as observed for primary-fruit growth in navel (14,52) and 'Valencia' (10) sweet oranges. The stage I of development of the secondary fruit lasted until late May, approximately 8 weeks after anthesis. Growth of the secondary fruit during stage I was due primarily to cell divisions in the peel. The stage II lasted from late May until early October and was characterized by rapid cell enlargement in the segments of the secondary fruit. The stage III consisted of slow growth of the secondary fruit accompanied by a gradual change of the color of the peel from green to orange. The beginning of the cell-enlargement stage (stage II) of the secondary fruit occurred approximately 2 weeks after that of the primary fruit. The overall developmental pattern of the secondary fruit, however, was similar to that of the primary fruit.

Fig. 1.9. Changes in volume of the primary and secondary fruit, and in diameter of the stylar-end aperture from anthesis until legal maturity of the fruit in navel orange, 1980. Each value represents the mean of a 30-fruit sample.


PRIMARY FRUIT
Volume (cm3)
250
150
50
0
A
M
J
J
A
S
O
SECONDARY FRUIT
Volume (cm3)
5
2
0
A
M
J
J
A
S
O
STYLAR-END APERTURE
Diameter (mm)
9
6
3
0
A
M
J
J
A
S
O

The aperture at the stylar-end of the primary fruit was not noticeable until mid-May, but then developed rapidly and reached final diameter in early August (Fig. 1.9).

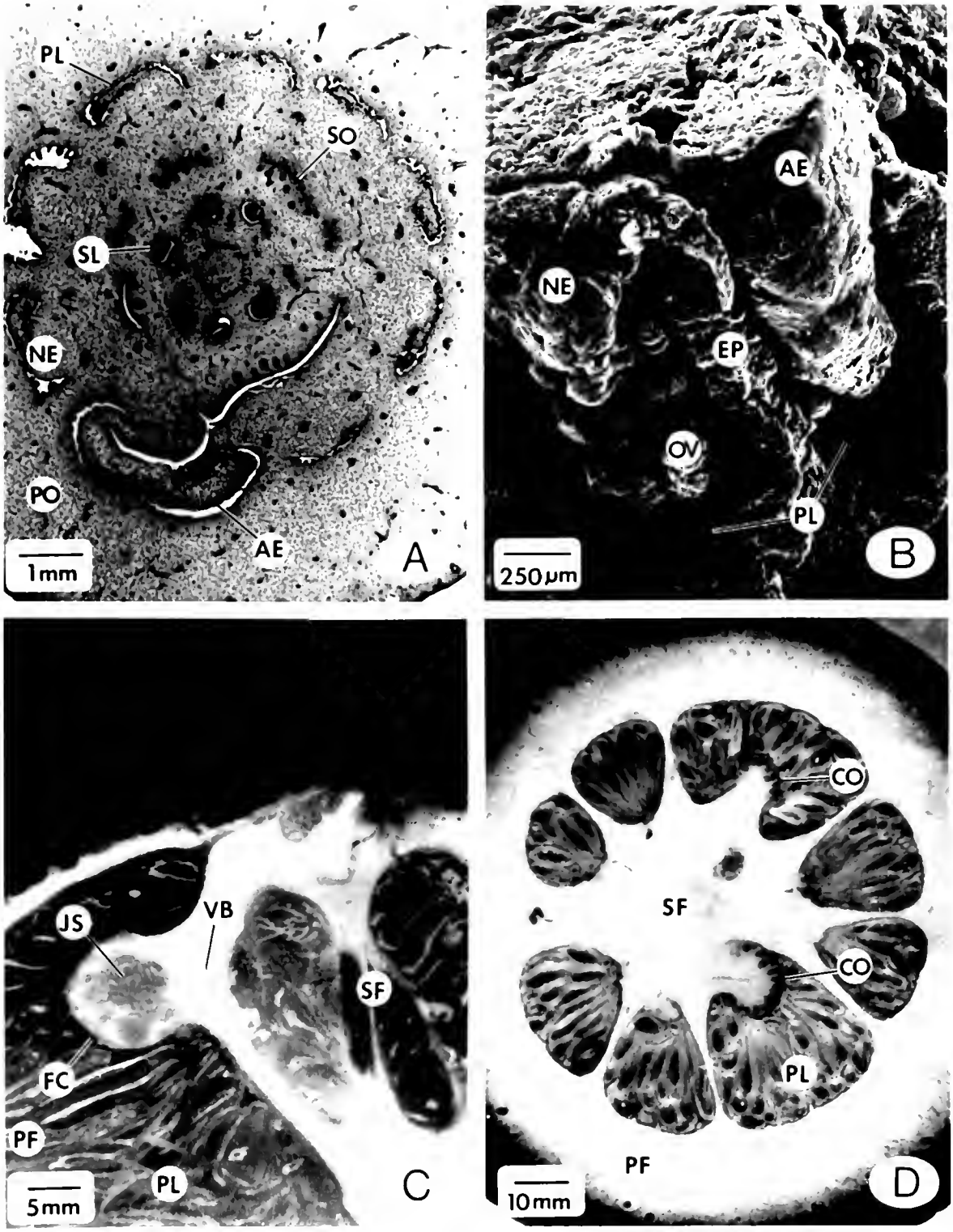
Secondary-Fruit Protrusions

Three types of tissue protrusions extended from the secondary fruit into primary-fruit locules, namely, abnormal placentae, free secondary carpels and secondary-carpel outgrowths. An abnormally large placenta developed and almost filled the locular cavity in a few instances (Fig. 1.10 A,B). These abnormally large placentae established tissue connections with secondary-fruit carpels (Fig. 1.10 A) but could not be distinguished from other types of protrusions at later stages of primary-fruit development.

Another type of protrusion resulted from free secondary carpels extending into adjacent primary-fruit locules (Fig. 1.10 C). These protrusions had juice sacs and carpellary vascularization and were found in all stages of primary-fruit development. Development of free secondary carpels caused separation of the primary-carpel margins in affected locules.

The most common type of protrusion resulted from secondary-carpel rind outgrowths extending into the primary-fruit locules (Fig. 1.10 D). These protrusions also caused separation of the primary-carpel margins as they extended into the locules and frequently established direct contact

Fig. 1.10. Tissue protrusions into primary-fruit locules of navel orange. A-B, at 20 days after anthesis: A, abnormally large placenta in a transection of the primary and secondary ovaries, and B, normal and abnormal placentae; C, free secondary carpel in median longisection of 60 mm-diameter fruit; D, secondary-carpel rind outgrowths in transection at the base of secondary fruit in the primary-fruit stylar-end. AE, abnormal placenta; CO, secondary-carpel outgrowth; FC, free secondary carpel; JS, juice sacs; NE, normal placenta; OV, ovule; PF, primary fruit; PL, primary-fruit locule; PO, primary ovary; SF, secondary fruit; SL, secondary-fruit locule; SO, secondary ovary; VB, vascular bundle.


PL
SO
SL
NE
PO
AE
1mm
A
AE
NE
EP
OV
PL
250µm
B
JS
VB
SF
FC
PF
PL
5mm
C
CO
SF
CO
PL
PF
10mm
D

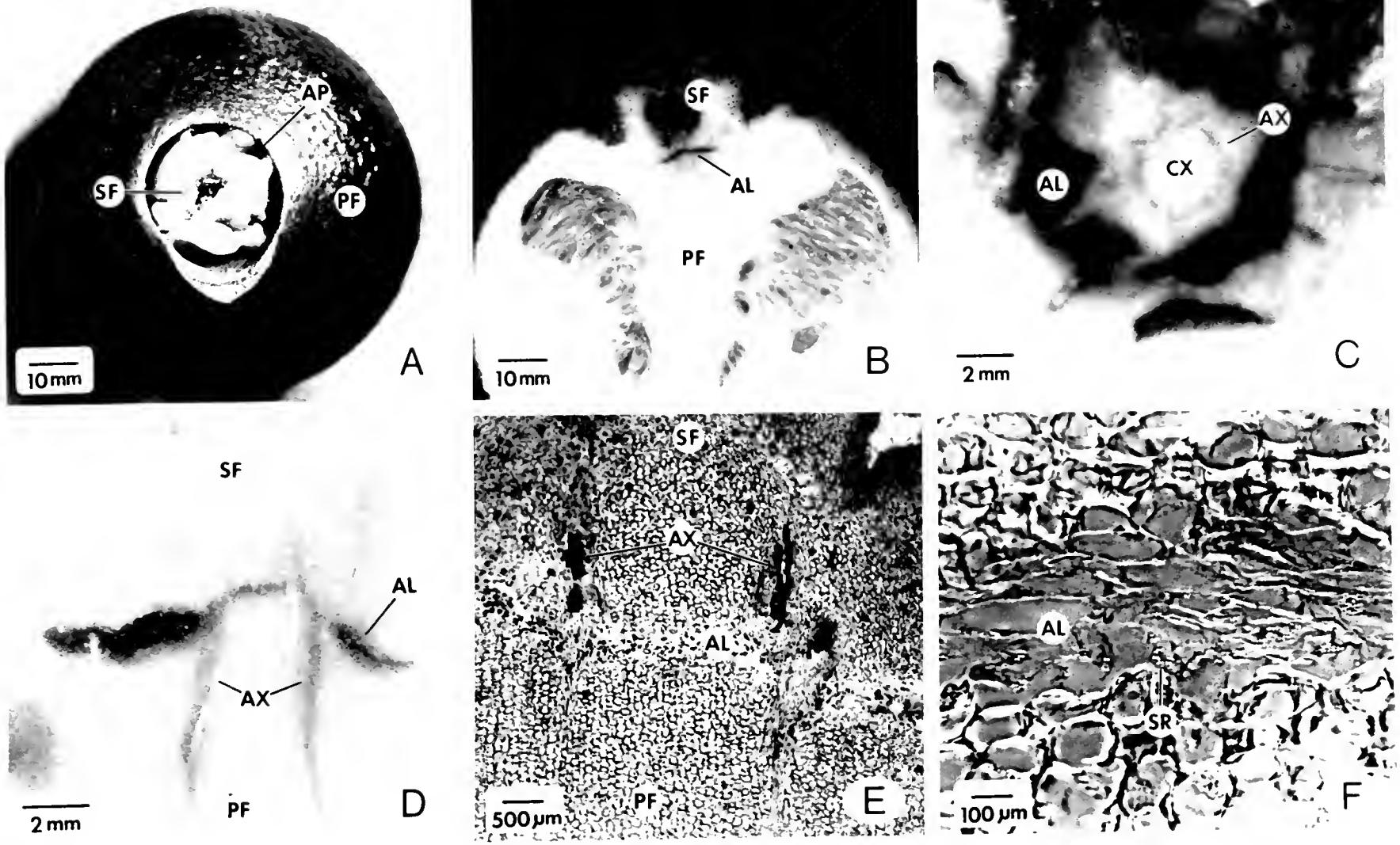
between a locular cavity and the outside of the primary fruit. Secondary-carpel rind outgrowths into the locules of primary fruit were found at all stages of primary-fruit development. These protrusions have been linked to stylar-end decay (77).

Secondary-Fruit Abscission

An abscission layer was detected in the central axis at the base of the secondary ovary. The abscission zone was not easily distinguishable anatomically from adjoining tissues prior to abscission, as is true for the primary-fruit abscission zone (19,124). The abscission zone at the base of the secondary fruit was composed of mesocarp-like tissue with parenchyma cells surrounding xylem and phloem of the axial vascular bundles, and parenchyma cells of the pith. Parenchyma cells in the abscission zone of the secondary fruit were thin-walled, isodiametric, slightly smaller, and had fewer intercellular spaces than adjacent parenchyma cells. Xylary and phloic fibers were less numerous in vascular bundles in the abscission zone than on either side of it.

Secondary-fruit abscission was observed only in fruit with symptoms of secondary-fruit yellowing (Fig. 1.11 A,B), which results in fruit drop of navel orange from early June until early August (77,78,79). Abscission or separation of

Fig. 1.11. Secondary-fruit abscission in navel orange. A-F, fruit 45 to 60 mm in diameter (June-July) with secondary-fruit yellowing: A, secondary-fruit yellowing symptoms, B, median longisection, C, transection of the abscission layer, D, median longisection of the abscission layer, E, longisection of the abscission layer, and F, starch grains in a longisection of the abscission layer. AL, abscission layer; AP, stylar-end aperture; AX, axial vascular bundle; CX, central axis; PF, primary fruit; SF, secondary fruit; SR, starch grains.


AP
SF
PF
10mm
A
SF
AL
PF
10mm
B
AX
CX
AL
2 mm
C
SF
AL
AX
2 mm
PF
D
SF
AX
AL
500µm
PF
E
AL
SR
100 µm
F

cells commenced in parenchyma cells that surround the axial vascular bundles in the abscission layer, and extended into parenchyma cells of the pith. Parenchyma cells of the abscission zone underwent cytolysis, which resulted in development of a gelatinous layer initially around the axial bundles only (Fig. 1.11 C), but later extending into the pith (Fig. 1.11 D). Abscission at the base of the primary fruit, however, starts in the pith and progresses outwardly to the cortex and epidermis (19,124).

The abscission layer located at the base of the secondary fruit is typically 2 to 3 cells thick and approximately 5 to 10 mm in diameter (Fig. 1.11 E). Parenchyma cells in the abscission layer of the secondary fruit possessed numerous starch grains during abscission, as reported for other abscission layers in citrus (19,124). Starch grains were present also in parenchyma cells on both sides of the separation layer and in the lysogenous intercellular spaces created by the disintegration of cells after abscission of the secondary fruit (Fig. 1.11 F).

Secondary-fruit abscission resulted in the decay of the secondary fruit, but the latter did not fall since its mesocarp is typically fused to the mesocarp of the primary fruit. The decay of the secondary fruit frequently extended into the primary fruit and caused the drop of the latter. The primary fruit remained unaffected in some instances, however, and a protective layer of suberized cells (i.e., a periderm) was formed on both sides of the secondary-fruit

abscission zone and sealed off the exposed tissues of the primary fruit. The periderm in the protective layer consisted of a phellogen which produced a phelloderm (2 to 5 layers of thin-walled parenchyma cells) and a phellem. The latter was composed of small cork cells and thick-walled, isodiametric parenchyma cells. As a result of the formation of the protective layer, the secondary fruit remained in the stylar-end cavity of the primary fruit as a mummified structure and the primary fruit did not fall. These primary fruit, however, frequently were affected by stylar-end decay later on.

Summary

The secondary fruit (navel) of navel orange develops as a whorl of secondary-carpel primordia within the primary-carpel whorl around the floral meristem when the flower bud is about 1.5 to 2 mm in length. A complete secondary pistil with fused ovary but separate styles and stigmas develops entirely within the primary pistil before anthesis. Stigmas and styles are not as distinct in secondary carpels as in primary ones. Secondary styles usually abscise following abscission of the primary style, but may simply break off.

Secondary and primary fruit have similar sigmoid growth curves, except the onset of the cell-enlargement stage in the former lags approximately 2 weeks.

Three types of tissue protrusions from the secondary fruit into primary-fruit locules were detected, namely, abnormal placentae, free secondary carpels and secondary-carpel outgrowths. Abnormal placentae almost fill the primary-fruit locules and are continuous with secondary-fruit rind tissue. Free secondary carpels and secondary-carpel outgrowths extend into the primary-fruit locules and cause separation of carpel margins. Secondary-carpel outgrowths were the most common type of protrusion and related to stylar-end decay.

An abscission layer is present in the central axis of the primary fruit at the base of the secondary ovary. The abscission layer is anatomically indistinguishable from adjacent tissues before abscission of the secondary fruit. Parenchyma cells in this layer have large numbers of starch grains during abscission. Secondary-fruit abscission was detected only during late spring and early summer in fruit affected by secondary-fruit yellowing.

CHAPTER II

PHYSIOLOGICAL STUDIES ON SECONDARY-FRUIT YELLOWING OF NAVEL ORANGE

Introduction

Lower yields of navel orange trees have been attributed to excessive drop of reproductive structures (23,30,67,73, 119). Previous research on fruit drop of navel orange in Florida included only the fruit-set period from bloom until early June (26,68,125). However, summer drop between early June and early August has been significant in 3 out of 4 seasons from 1978 to 1981 with up to 101 fruit, or 15% of the crop, falling per tree in a single season, virtually all with symptoms of secondary-fruit yellowing (78).

Secondary-fruit yellowing consists of an early discoloration of the secondary fruit, or navel, while the primary fruit remains sound. Secondary decay-organisms and insects are observed only at late stages of secondary-fruit yellowing. The problem appears to result from secondary-fruit abscission (77).

Fruit abscission is typically promoted by ethylene (82, 116), but inhibited by auxins (29,39,53,113). Secondary-fruit yellowing can be induced by ethylene treatment (112), but effects of auxins on the development of the disorder have not been studied.

Water stress may cause abscission of young citrus fruit, particularly those of navel orange (23,26,119). Carbohydrate levels in the plant also may influence young fruit drop in citrus (59,60,74). Neither water nor carbohydrate status have been studied, however, in relation to summer drop of navel orange.

Secondary-fruit yellowing was induced by fruit-stem ringing and effects of time of ringing, distance and number of leaves between the ring and a fruit and application of 2,4-dichlorophenoxyacetic acid were studied. Levels of cellulase and ethylene in the abscission zone of the primary and secondary fruit, and fruit removal force were determined at different stages of secondary-fruit yellowing. In addition, the role of leaf abaxial diffusive resistance, xylem water potential and nonstructural carbohydrates in secondary-fruit yellowing was investigated.

Literature Review

Secondary-Fruit Yellowing

Secondary-fruit yellowing (SFY) of navel orange occurs during late spring and early summer when fruit are approximately 35 to 65 mm in diameter (77,79). The secondary-fruit rind exposed through the aperture at the stylar end of the primary fruit becomes yellow, but the secondary fruit decays only at advanced stages of SFY. The primary fruit remains green and sound during development of SFY, but finally becomes yellow at the stylar-end and abscises.

Insects and microorganisms do not damage secondary fruit at early stages of SFY (77,79). Later, however, secondary fruit decays and attracts sap beetles (Coleoptera, Nitidulidae). Southwick et al. (112) isolated fungi from decaying fruit affected by SFY and reinoculated healthy fruit in the presence or absence of ethylene. They concluded fungi and ethylene are related to SFY but do not appear to be causal factors. Moreover, SFY was not controlled by benomyl or malathion sprays (77,78).

Secondary fruit of fruit with early symptoms of SFY were found to have separated from the primary fruit through a zone at the base of the secondary ovary resembling an abscission zone. Separation of the secondary fruit in fruit

affected by SFY in the absence of primary insect or fungi damage suggests the possibility of a physiological cause for summer drop of navel orange in Florida. Fruit affected by SFY produce large amounts of ethylene (112), but other physiological aspects of SFY and secondary-fruit abscission have not been studied.

Fruit Abscission

The abscission process. Abscission is brought about by the loss of cementing capability of cell wall material in the separation layer, followed by mechanical breakage of nonliving vascular elements and the epidermis (11,92,116). Leopold (72) divided the abscission process into 5 stages. Differentiation of an abscission zone usually occurs prior to the time of most active fruit enlargement. A holding stage follows in which no weakening occurs in the abscission zone. This stage usually lasts for the functional life of the subtending organ. The holding stage is followed by stages of structural weakening of the abscission zone, separation, and healing. Not all these stages, however, are essential, e.g., the abscission zone at the base of citrus fruit is not clearly differentiated, and no healing occurs of the exposed surface on the plant after fruit abscission (92,124).

Abscission of a citrus fruit begins with the swelling of cell walls in the separation layer followed by cell wall

dissolution and exudation of cellular contents into the break created by separation. The separation layer becomes gelatinous with no distinguishable intact cell structures, except for tracheary elements and the epidermis (124).

Cytolysis during abscission has been linked to increased enzymatic activity. Wilson and Hendershott (124) detected significant demethylation of pectins in the fruit abscission zone, which produced a distinct band of cells low in methylated pectins, the separation layer. Pectins act as cementing substances between cells and are linked by calcium and magnesium ions (101). Loss of these ions from cells in the separation layer during abscission indicates the involvement of pectinases, e.g., polygalacturonase (13, 87,97,114,124).

Biggs (13) related the swelling of cell walls in separation layers of citrus to a decrease in cross-linkage among polymers and endobreaking of polymers. He concluded that cellulases are involved in the abscission of citrus fruit, as reported previously (1,93,97). Cellulase activity has been related to reduced fruit removal force in citrus (42,63). Other enzymes—peroxidase, dehydrogenase, and acid phosphatase—also have been associated with the abscission process (11,72).

Accumulation of starch has been reported in cells of the separation layer prior to abscission in several plants (11) including citrus (19,124). No explanation for this phenomenon has been proposed. Large numbers of accumulated

starch grains which remain intact throughout abscission eventually appear in the intercellular spaces formed after cell separation (19,124).

Growth regulators. Growth regulators are intimately involved in fruit abscission (82,116). Abscission can be inhibited or promoted by auxins but is inhibited in most cases (13,45,72,116). Abscission promotion by auxins appears to result from the induction of high levels of ethylene, especially at high concentrations of auxin (116). The delay in abscission by auxins occurs principally through inhibition of passage out of the holding stage (72). Progression from the holding into the structural weakening stage apparently is due to a decline in endogenous auxin levels (72,116). Inhibition of abscission by auxins has been related in some instances to decreased activity of cell wall macerating enzymes such as cellulases and polygalacturonase in the abscission zone (13,41,97,99). Auxin sprays prevent fruit abscission in several crops (11), including citrus (5, 29,39,83,84,89). The effect, however, is dependent upon concentration and species or cultivar.

Ethylene generally has a stimulating effect on abscission (3,72,82,116), and may be the major regulator of the process, with other factors acting through inhibition or promotion of ethylene (116). Ethylene is a strong promotor (24,25,92) as well as a natural product of citrus abscission (92). Ethylene affects abscission primarily at the stage of

structural weakening when senescence has commenced in the tissues distal to the separation layer (72). Abscission in young citrus fruit is not promoted or hastened by ethylene or ethylene-generating compounds in some instances (53,64, 85). This has been attributed to the lack of tissue sensitivity (53,75) and high levels of auxin (85). A decline in auxin levels with aging increases tissue sensitivity to ethylene and allows abscission. Ethylene appears to promote abscission by increasing the activity of cell wall hydrolases, particularly cellulases (3,13,43,97) and polygalacturonase (43,99), and by inducing senescence (1,2,3).

Gibberellins, abscisic acid and cytokinins also influence abscission (27,72,96,116). Gibberellins seem to promote abscission when applied to abscission zones (116) or entire citrus trees (68), but prevent abscission when individual fruit are treated (21,68). Abscisic acid generally enhances fruit abscission (3,11,116), probably by promoting senescence of distal tissues (72) and stimulating ethylene and cellulase biosynthesis (3,72). Cytokinins weakly inhibit abscission and are thought to act like auxins by delaying the completion of the holding stage or slowing down senescence (72,82,116).

Hormone interaction and balance have been suggested to control abscission (3,18,116). Most hypotheses involve an auxin-ethylene balance, because the effects of these hormones on abscission are better known (18).

Materials and Methods

Plant Material

Navel orange (Citrus sinensis (L.) Osbeck) trees on sour orange (Citrus aurantium L.) rootstock, planted in 1957 were used for experiments in 1980. Similar trees, planted in 1969, were used in 1981 and 1982. Trees were located near Eustis, in the north central citrus region of Florida. Experimental trees received general care, including irrigation, typical of groves with fruit destined for the the fresh-fruit market.

Induction of Secondary-Fruit Yellowing

Secondary-fruit yellowing (SFY) was induced by fruit-stem ringing from late May until late July. This consisted of the removal of a 1 cm ring of bark from the fruit stem, 5 to 10 cm from the fruit, and excision of all leaves between the ringed area and the fruit. Secondary-fruit yellowing was also achieved without leaf removal so long as the ringed area was within 10 cm of the fruit.

Application of 2,4-D and Ethephon

Applications of 2,4-dichlorophenoxyacetic acid (2,4-D) were made to whole trees, whole fruit, or to parts of fruit

or fruit stem. Whole-tree applications consisted of 20 ppm 2,4-D sprays in 55 liters of solution on April 19 or May 6, 1982. Whole-fruit applications consisted of 4 to 8 biweekly sprays of 10 ppm 2,4-D before or after fruit-stem ringing. Applications of 2,4-D to the ringed area or the stylar-end of stem-ringed fruit were made at the time of ringing as a 1000 or 2000 ppm slurry in lanolin paste. Control fruit received lanolin without 2,4-D. Control fruit and trees were not sprayed. Applications of ethephon (2-chloroethyl-phosphonic acid) to the stylar-end of unringed fruit were made as a 10 sec dip in a 300 ppm solution.

Ethylene, Cellulase and Fruit Removal Force

Ethylene concentration and cellulase activity in the abscission zone at the base of the primary and secondary fruit, and fruit removal force (FRF) were determined at different stages of SFY. Secondary-fruit yellowing stages were 1, green (healthy secondary fruit), 2, early (slight discoloration of the secondary fruit), 3, moderate (yellowing but no secondary decay), 4, advanced (yellowing and secondary decay), and 5, very advanced (advanced decay and yellowing symptoms spreading to primary-fruit areas around the secondary fruit). Ten to 20 fruit were collected at each stage and determinations made on the same day. Twenty to 40 fruit were analyzed daily. Determinations were

made in 7- to 10-day periods during the peak of SFY incidence in late June to early July in 1981 and 1982.

Ethylene concentration was determined by gas chromatography on samples taken with disposable syringes from central axis tissues which included either the primary- or the secondary-fruit abscission zone. Fruit were cut in half transversally under water, a hypodermic needle was placed either into the central axis of the stem half toward the primary-fruit abscission zone or of the stylar half toward the secondary-fruit abscission zone, and a 1 ml gas sample withdrawn. Samples were injected into a Hewlett-Packard 5710A gas chromatograph equipped with a flame ionization detector and a stainless steel 1 m x 6.35 mm ID column packed with activated alumina. Flow rate was 75 ml per min using nitrogen as a carrier gas. Oven temperature was 100 C.

Cellulase activity was determined by viscometry (76). Samples of 30 to 50 mg of tissue containing the abscission zone plus a small amount of adjacent tissue were excised with a scalpel. Cellulases were extracted by grinding the tissue sample with mortar and pestle in 2 ml of 20 mM sodium phosphate buffer (pH 6.5) and centrifuging at 10,000 g for 10 min (63). Extraction was done at temperatures under 4 C. Cellulase activity was assayed in the supernatant by measuring the reduction in viscosity of a 1.2% solution of carboxymethylcellulose (CMC) type 7H3SF (Hercules Powder Co., Wilmington, Del.) in 20 mM sodium phosphate buffer

(pH 6.5) (98). The assay mixture consisted of 0.4 ml of CMC solution and 0.2 ml of the enzyme extract. Viscosity was determined by measuring drainage time of the assay mixture through a calibrated portion of a 0.1 ml pipette between the 0.00 and 0.03 ml marks (76). The change in viscosity from time zero (time of mixing enzyme and substrate) and 1 to 2 hours of incubation at 24 C was used to measure enzyme activity. Data were converted to units of cellulase activity per abscission zone and time (hour) (7,76).

Fruit removal force (FRF) was measured by clamping the fruit-explant stem to a Chatillon strain gauge (John Chatillon & Sons, New York, NY) and applying tension manually by pulling the fruit until separation from the stem occurred. The FRF was the maximum force required for separation.

Water and Carbohydrate Status

Secondary-fruit yellowing was induced by fruit-stem ringing with no removal of leaves between the ringed area and the fruit. Control fruit were not ringed. Four fruit were ringed per replication with 5 replications per treatment.

Branches were sawed halfway at 3 locations approximately 5 cm apart on alternate, opposite halves of the axis in an attempt to restrict xylem translocation

although phloem translocation was also restricted. Branch scoring consisted of a single knife-cut made through the bark around the main axis in an attempt to restrict phloem translocation only. Branches sawed or scored, and control untreated branches were ca. 2.5 cm in diameter and held an average of 13 fruit. Ten branches were used per treatment.

Trunk girdling consisted of a single knife-cut through the bark around the trunk at 10 to 20 cm above the bud union. Control trees were not girdled. Five single-tree replications were used per treatment.

Leaf abaxial diffusive resistance, xylem water potential, and total nonstructural carbohydrates were determined on fully expanded spring-flush leaves after induction of SFY by fruit-stem ringing, and after branch sawing or scoring. Total nonstructural carbohydrates of leaves were also determined after trunk girdling. Leaf measurements were made on 1 subtending leaf (closest to the fruit) per ringed fruit, 2 to 3 leaves per sawed or scored branch, or 10 leaves per girdled tree. Leaves from treated branches or trees were collected from fruitless shoots located randomly on the outside of the canopy at 1 to 2 m-height.

Leaf abaxial diffusive resistance was measured with a LI-COR LI-1600 steady state porometer. Leaf xylem water potential was obtained using the Scholander pressure chamber technique (103). Leaf abaxial diffusive resistance and xylem water potential were measured between 12 and 2 PM. Total

nonstructural leaf carbohydrate level was determined by treating leaf extract suspensions with invertase, amyloglucosidase and takadiastase (81). Enzyme-digested suspensions were then analyzed for reducing sugars according to a modified Nelson copper reduction test (90,108).

Evaluation of Secondary-Fruit Yellowing

Secondary-fruit yellowing incidence was evaluated after fruit-stem ringing and growth regulator treatments through weekly inspection of treated fruit for 25 to 45 days after ringing. Total number of SFY-affected fruit was obtained for the entire period and expressed as % of treated fruit. Secondary-fruit yellowing after branch sawing or scoring was estimated by counting the number of affected fruit from June 10 until August 6 and expressed as % of the initial number of fruit per branch.

Experimental Designs

Completely randomized or randomized complete block designs were used. Mean separation was done by Duncan's multiple range test, utilizing Kramer's adaptation for experiments with a variable number of replications (65).

Results and Discussion

Maximum SFY was obtained from fruit-stem ringing on May 26 and 28 in 1982 (Table 2.1). The % secondary-fruit yellowing was lower on dates prior to or after these, with no SFY induction achieved on May 6 or August 6. Natural SFY occurred from early June until early August with a peak during late June or early July. It is possible that the time of peak natural induction of SFY was also during late May in 1982. Similar results were obtained in 1981, with maximum SFY induction obtained from fruit-stem ringing performed on June 15 (data not shown), or about 2 weeks later than in 1982. Full bloom and petal fall in 1981 also occurred 2 to 3 weeks later than in 1982. As a result, fruit size at the time of peak SFY was comparable in both seasons and ranged from 45 to 55 mm in diameter.

Fruit-stem ringing of navel orange induced higher ethylene levels in the stylar-end of the fruit 7 days after ringing, accompanied by SFY, which was followed by fruit drop (Table 2.2). Although ethylene is produced in plant tissues after wounding (24,82,116), the responses to fruit-stem ringing probablywere not due to high ethylene levels in treated fruit. No increase in ethylene concentration was detected after fruit-stem ringing when SFY was inhibited by 2,4-D application (Table 2.2). In addition, ringing did not increase ethylene levels or produce SFY symptoms or fruit drop in 'Hamlin' orange (Table 2.2), which typically does

Table 2.1. Secondary-fruit yellowing (SFY) of navel orange as affected by date of induction by fruit-stem ringing, 1982.

Date of fruit-stem ringing	SFY (% of treated fruit)[z]
May 6	0c[y]
May 21	60b
May 26	90a
May 28	90a
June 3	65b
August 6	0c

[z]Ten to 30 fruit treated per date. Evaluation of SFY was done until 25-40 days after ringing.

[y]Unlike letters indicate significant differences by Duncan's multiple range test, 1% level.

Table 2.2. Fruit ethylene, secondary-fruit yellowing (SFY), and fruit drop of navel and 'Hamlin' orange following fruit-stem ringing and ethephon (ETH) or 2,4-dichlorophenoxyacetic acid (2,4-D) stylar-end treatments, 1982.

Treatments[z]	Fruit ethylene (ppb)[y]		SFY[x] (%)	Fruit drop[x] (%)
	Stem-end	Stylar-end		
Navel				
No ringing	70a[w]	74b	0c	0c
Ringing	96a	347a	90a	90a
No ringing + ETH	—[v]	—	65b	65b
Ringing + 2,4-D	88a	126b	0c	0c
'Hamlin'				
No ringing	71a	97b	—	—
Ringing	97a	82b	—	—

[z]Two to 6 fruit per replication, 5 replications per treatment, applied on May 28. Stylar-end treatments consisted of a 300 ppm ethephon dip or an application of lanolin paste containing 1000 or 2000 ppm 2,4-D.

[y]Sampled from the central axis at the primary-fruit abscission zone in the stem-end, and at the secondary-fruit abscission zone in the stylar-end, 7 days after treatment, 7 fruit per treatment.

[x]Evaluated until July 16. Fruit drop followed SFY in all instances and both are expressed as % of treated fruit.

[w]Unlike letters within columns indicate significant differences by Duncan's multiple range test, 1% level.

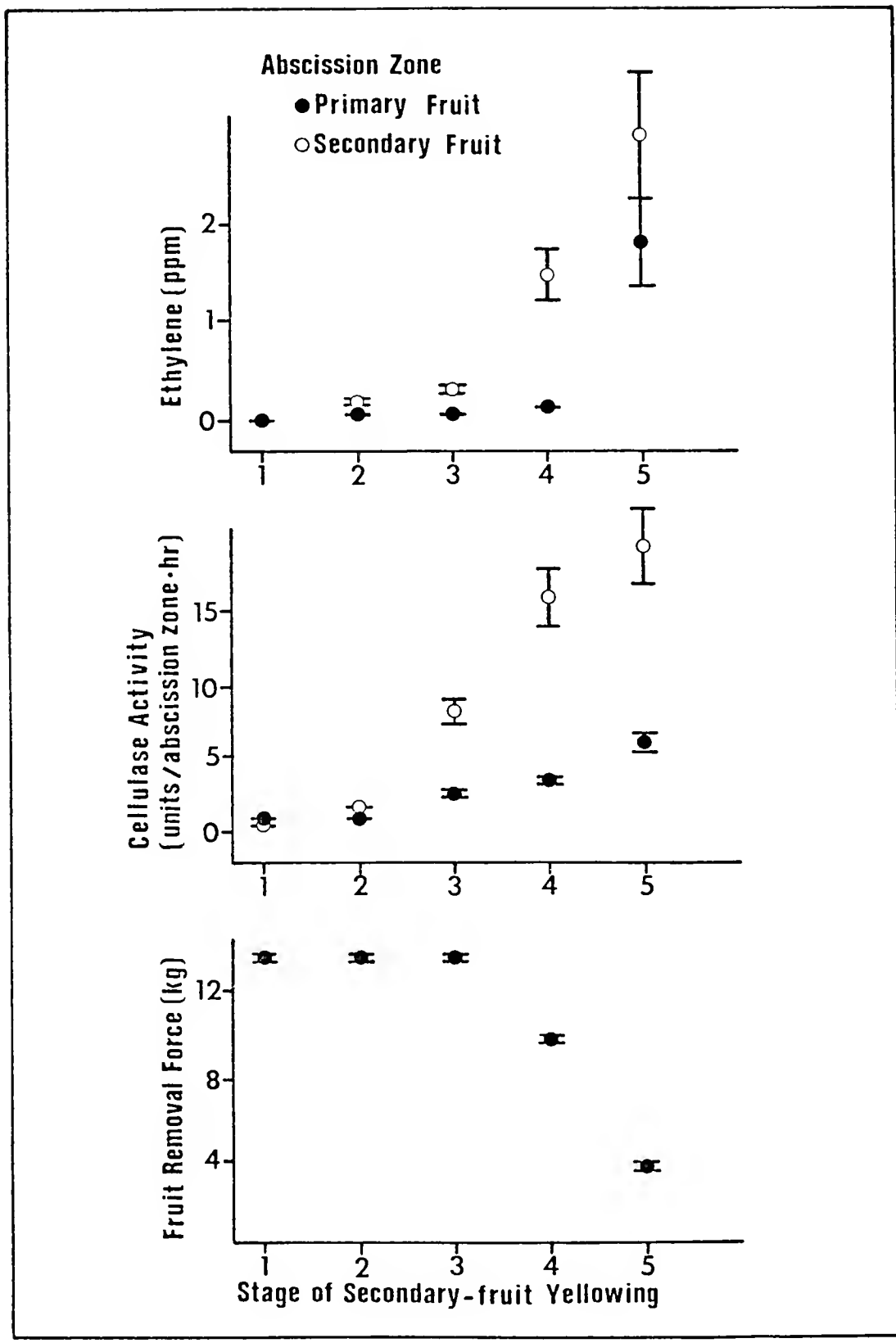
[v]Not determined or not applicable.

not have a secondary fruit (51). Fruit growth, however, ceased after fruit-stem ringing in both navel and 'Hamlin' (data not shown). It appears that higher fruit ethylene levels after fruit-stem ringing in navel orange occur after rather than before secondary-fruit abscission and SFY.

Fruit abscission is typically promoted by ethylene (116), but inhibited by auxins (29,39,53). Navel oranges treated with 2,4-D at the stylar-end did not respond to fruit-stem ringing. Dipping the fruit stylar end in a 300 ppm ethephon solution for 10 sec, however, did result in SFY, followed by fruit drop (Table 2.2), as previously reported (109,112).

Ethylene levels and cellulase activity began to increase in the abscission zone of the secondary fruit during stage 2 of SFY, when visible discoloration was first detected (Fig. 2.1). It appears that secondary-fruit abscission preceded development of SFY symptoms and subsequent invasions by sap beetles and decay organisms. Conversely, high levels of ethylene and cellulase activity, as well as reduced fruit removal force, were observed in the primary-fruit abscission zone only at advanced stages of SFY (Fig. 2.1). Fruit with SFY symptoms produce high amounts of ethylene (112). Ethylene induces abscission probably by stimulating cellulase activity (3,13) which in turn reduces fruit removal force (42,63). It appears that secondary-fruit abscission precedes and is the cause of primary-fruit abscission during summer drop.

Fig. 2.1. Ethylene, cellulase activity, and fruit removal force in the abscission zone at the base of the primary and secondary fruit in relation to stage of secondary-fruit yellowing of navel orange, 1982. Each value represents the mean of a 10- to 20-fruit sample ± SE. Stages of secondary-fruit yellowing were 1, green (healthy secondary fruit, SF), 2, early (slight discoloration of SF), 3, moderate (yellowing but no decay), 4, advanced (yellowing with decay), and 5, very advanced (yellowing with advanced decay).


Abscission Zone
Primary Fruit
Secondary Fruit
Ethylene (ppm)
0
1
2
Cellulase Activity
(units/abscission zone·hr)
0
5
10
15
Fruit Removal Force (kg)
4
8
12
1
2
3
4
5
Stage of Secondary-fruit Yellowing

Water stress has been implicated in the abscission of young citrus fruit, particularly those of navel orange (23, 26,50,122). Carbohydrate levels in the plant also may influence fruit drop during the fruit set period (59,60,74). Fruit-stem ringing resulted in no change in leaf abaxial diffusive resistance, xylem water potential or total nonstructural carbohydrates but did induce secondary-fruit yellowing (Table 2.3). Conversely, branch sawing and scoring treatments resulted in changes in leaf water and nonstructural-carbohydrate status during the SFY-induction period in late May but did not influence summer fruit drop in 1981 (data not shown) and in 1982 (Table 2.4). In addition, trunk girdling in early May increased the levels of nonstructural carbohydrate in the leaves but had no effect on summer fruit drop or yield (Table 2.5 and Chapter III, Table 3.9). Apparently, the water and nonstructural-carbohydrate status of fully expanded spring-flush leaves are not associated with SFY.

Fruit-stem ringing at 5 to 10 cm from the fruit, with or without leaf removal, resulted in induction of SFY in 80 to 100% of treated fruit (Table 2.6). Significantly less SFY induction was achieved as the distance of the ringed area from the fruit, and the number of leaves above the ringed area increased. Furthermore, removal of all leaves up to 30 to 45 cm from the fruit in the absence of fruit-stem ringing did not induce SFY (Table 2.6). Secondary-fruit yellowing, therefore, can be induced when bark-translocation

Table 2.3. Leaf abaxial diffusive resistance(ADR), xylem water potential (Ψ_x), total nonstructural carbohydrates (CHO), and secondary-fruit yellowing (SFY) of navel orange as affected by fruit-stem ringing, 1982.

Treatments[z]	Leaf measurements[y]			SFY[x] (%)
	ADR(s cm^{-1})	Ψ_x(MPa)	CHO(mg g^{-1} d.w.)	
Control	0.19	-1.55	51.9	0
Ringing	0.32ns	-1.39ns	41.5ns	65**

[z]Four fruit per replication, 5 replications per treatment, applied on June 3.

[y]One fully expanded spring-flush leaf subtending each fruit. ADR and Ψ_x were evaluated on June 10 and CHO on June 29.

[x]Evaluated until July 16 as % of treated fruit.

ns,**Nonsignificant (ns) or significant at 1% level (**) by a t-test.

Table 2.4. Leaf abaxial diffusive resistance (ADR), xylem water potential (Ψ_x), total nonstructural carbohydrates (CHO), and secondary-fruit yellowing (SFY) of navel orange as affected by branch sawing or scoring, 1982.

Treatments[z]	Leaf measurements[y]			SFY[x] (%)
	ADR ($s \cdot cm^{-1}$)	Ψ_x (MPa)	CHO (mg g^{-1} d.w.)	
Control	4.31b[w]	-1.30b	98.2a	7.8a
Sawing	8.58a	-1.59a	72.5b	7.6a
Scoring	4.04b	-1.34b	115.6a	9.3a

[z]Ten branches (2.5 cm-diameter) per treatment, 13 fruit per branch, applied on May 8. The main axis was sawed halfway transversally at 3 locations 5 cm apart. The cuts were made on alternate opposite halves of the axis in an attempt to restrict xylem translocation although phloem translocation was also restricted. Scoring consisted of a single knife-cut through the bark around the main axis in an attempt to restrict phloem translocation.

[y]Determined on May 21 using 2 to 3 fully expanded spring-flush leaves per branch.

[x]From June 10 to August 6. Expressed as % of initial number of fruit per branch.

[w]Unlike letters within columns indicate significant differences by Duncan's multiple range test, 5% level.

Table 2.5. Leaf total nonstructural carbohydrates (CHO) of navel orange as affected by trunk girdling, 1982.

Treatments[z]	CHO (mg g^{-1} d.w.)[y]		
	May 15	May 21	June 29
Control	106	98	65
Trunk girdling (May 6)	106ns	99ns	72*

[z]Trunk girdling consisted of a single knife-cut through the bark around the trunk 10 to 20 cm above the bud union.

[y]Ten fully expanded spring-flush leaves per tree and 5 trees per treatment were used at each date.

ns,*Nonsignificant (ns) or significant at 5% level (*) by a t-test.

Table 2.6. Secondary-fruit yellowing (SFY) of navel orange following fruit-stem or branch ringing and leaf removal treatments, 1980-1982.

Treatments[z]	Number of leaves between ring and fruit	SFY (% of treated fruit)		
		1980	1981	1982
No ringing, no leaf removal	—[y]	0b[x]	0b	10c
No ringing, leaves removed from 30-45 cm of fruit branch	—	0b	10b	5c
Ringing at 5-10 cm, leaves removed	0	100a	80a	90a
Ringing at 5-10 cm, no leaf removal	2-3	90a	100a	—
Ringing at 15-20 cm, no leaf removal	6-15	—	—	60b
Ringing at 30-45 cm, no leaf removal	20-55	—	—	20c

[z]Four to 5 replications per treatment, 2 to 6 fruit per replication, treated from late May to early June and evaluated until 20 to 35 days later.

[y]Not applicable or not determined.

[x]Unlike letters within years indicate significant differences by Duncan's multiple range test, 5% level.

of leaf metabolites to the fruit is limited. Little SFY, however, results from fruit-stem ringing if enough leaves remain between the ringed area and the fruit, and sufficient leaf metabolites are supplied to the fruit. Brown (16) and Powell and Krezdorn (95) found that movement of ^{14}C-metabolites from the leaf to the fruit influenced citrus fruit development and young-fruit abscission.

Induction of SFY by fruit-stem ringing was prevented by applying 2,4-D to the ringed area, the stylar-end, or as whole-fruit sprays prior to or after ringing (Table 2.7). The only exception was with 2000 ppm 2,4-D applied to the ringed area. High auxin concentrations may promote abscission by stimulating ethylene biosynthesis (116). Secondary-fruit yellowing appears to result from secondary-fruit abscission; hence, reduction of SFY by 2,4-D may occur through inhibition of abscission of the secondary fruit. Auxins act as strong abscission antagonists in citrus (13,45,53) as well as in other plants (72,116). Leopold (72) suggested that the progression from the holding into the structural weakening stage of abscission is due to a decline in endogenous auxin levels, which increase tissue sensitivity to ethylene and allow abscission (85). It is possible that auxins or auxin-precursors are some of the bark-translocated leaf metabolites that would act similarly to exogenous 2,4-D. Ringing and leaf removal would limit auxin supply to the abscission zone of the secondary fruit, thereby allowing abscission and the development of SFY

Table 2.7. Secondary-fruit yellowing (SFY) of navel orange following fruit-stem ringing as affected by 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1982.

Fruit treatments[z]	SFY (% of treated fruit)[y]			
	Time and location of 2,4-D application			
	Preringing	Postringing	Ringed area	Stylar-end
No ringing, no 2,4-D	5b[x]	10b	0c	0b
Ringing, no 2,4-D	90a	87a	90a	100a
Ringing plus				
10 ppm 2,4-D	20b	27b	—	—
1000 ppm 2,4-D	—[w]	—	45b	0b
2000 ppm 2,4-D	—	—	70a	0b

[z]Four to 5 fruit per replication, 5 to 6 replications per treatment, applied on May 26 to 28. Sprays of 2,4-D were applied biweekly 4 to 8 times before (preringing) or after (postringing), or 2,4-D applied in lanolin paste on the ringed area (ringed area) or at the fruit stylar-end (stylar end) at the time of ringing. Ringing only treatments were either not sprayed or received lanolin without 2,4-D.

[y]Evaluated until July 16.

[x]Unlike letters within columns indicate significant differences by Duncan's multiple range test, 1% level.

[w]Not applicable.

symptoms. Accordingly, SFY which occurs during summer fruit drop may result from limited supplies of auxins or auxin-precursors to the developing fruit. Whole-tree 2,4-D sprays as early as full bloom were found to prevent SFY and significantly reduce summer fruit drop (Chapter III, Tables 3.7,3.8,3.9). In addition, attempts to induce SFY by fruit-stem ringing of fruit on 2,4-D sprayed trees were unsuccessful (Table 2.8). Sprays of 2,4-D were applied 3 to 5 weeks prior to fruit-stem ringing which made the fruit unresponsive to SFY induction.

Trunk girdling increases gibberellin levels (86,120) and fruit set in citrus (66,69,86). Trunk girdling of navel orange during early May resulted in increased leaf nonstructural-carbohydrate levels within 7 weeks of treatment (Table 2.5), but did not prevent SFY and summer fruit drop (Chapter III, Table 3.9), or induction of SFY by fruit-stem ringing (Table 2.8).

Summary

Induction of secondary-fruit yellowing (SFY) of navel orange by fruit-stem ringing or stylar-end ethephon treatment and its prevention by 2,4-D application suggest a physiological nature for the disorder. This contention is

Table 2.8. Secondary-fruit yellowing (SFY) of navel orange following fruit-stem ringing as affected by 2,4-dichlorophenoxyacetic acid (2,4-D) whole-tree application or trunk girdling, 1982.

Treatments[z]	Fruit-stem ringing[y]	SFY[x] (%)
No treatment	No	0b[w]
No treatment	Yes	89a
2,4-D on April 19	Yes	25b
2,4-D on May 6	Yes	15b
Girdling on May 6	Yes	80a

[z]Five to 10 trees per treatment. Sprays of 2,4-D were made at 20 ppm in 55 liters of solution per tree. Trunk girdling consisted of a single knife-cut through the bark around the trunk 10 to 20 cm above the bud union.

[y]Ringed on May 28.

[x]Evaluated until July 16 as % of treated fruit.

[w]Unlike letters indicate significant differences by Duncan's multiple range test, 1% level.

further supported by a cause-and-effect relationship between secondary-fruit abscission and SFY.

Secondary-fruit abscission precedes primary-fruit abscission and the levels of both ethylene and cellulase activity increase in the abscission zone of the secondary fruit prior to that of the primary fruit affected by SFY. Reduced fruit removal force also occurs only at advanced stages of SFY. Abscission of the primary fruit, which follows SFY, may result from increased ethylene levels after secondary-fruit abscission.

Induction of SFY by fruit-stem ringing was not associated with changes in ethylene levels of the fruit or the water and nonstructural-carbohydrate status of subtending leaves. Moreover, changes in water and nonstructural-carbohydrate status of leaves induced by branch sawing or scoring, or by trunk girdling had no significant effect on SFY incidence.

Fruit-stem ringing and leaf removal experiments indicated induction of SFY resulted from an interrupted supply of bark-translocated leaf metabolites to the fruit. Applications of 2,4-D to the whole tree, fruit, or to the ringed area on the fruit stem, before or after ringing, however, reduced induction of SFY from fruit-stem ringing.

CHAPTER III

GROWTH REGULATOR EFFECTS ON FRUIT DROP, YIELD AND QUALITY OF NAVEL ORANGE

Introduction

Fruit drop after the fruit-set period is significant (77,78,79), and may be responsible for lower yields of navel orange trees in Florida. Three major causes of fruit drop have been characterized, secondary-fruit yellowing, stylar-end decay, and fruit splitting, all of which have been associated with the morphology and anatomy of the stylar-end of the navel fruit, which encloses the secondary fruit (77, 79). Secondary-fruit yellowing which causes most of the summer fruit drop appears to result from abscission of the secondary fruit. Stylar-end decay and fruit splitting, the major causes of the summer-fall drop, more frequently affect fruit with larger stylar-end aperture.

Growth regulators influence fruit abscission (116). Auxin applications delayed or prevented preharvest fruit drop of sweet oranges in California (20,113) and of 'Pineapple' and seedling sweet oranges in Florida (39). In

addition, applications of 2,4-dichlorophenoxyacetic acid (2,4-D) in combination with gibberellic acid (GA) to individual flowers caused the peel of the primary fruit to extend over the secondary fruit resulting in smaller or absent stylar-end aperture in navel orange (68). Growth regulators, however, have not been examined in terms of their effects on summer and summer-fall fruit drop of navel oranges in Florida.

The objectives of this research were to study the effects of growth regulator sprays on summer and summer-fall fruit drop and on the external and internal characteristics of the fruit of navel orange in Florida.

Literature Review

Citrus Fruit Drop

Mature fruit usually develop from less than 1% of the reproductive structures produced by a sweet orange tree (33, 79). Shedding of reproductive structures takes place during all stages of development from flower buds to ripe fruit, typically occurring in cycles or waves (19,22,23,73,79). The first wave, bloom drop, starts at the flower-bud stage, has a peak during bloom and involves abscission of flower buds, flowers and young fruit (19,33). Abscission generally occurs

at the base of the pedicel. Most of the reproductive structures of sweet oranges fall during bloom drop. Erickson and Brannaman (33) reported 97.5% of the fruit of navel orange and 96% of those of 'Valencia' were shed before they reached 10 mm in diameter. Similarly, in Florida, 88.7% of navel orange ovaries were shed during the bloom drop period (79). The cause of bloom drop has not been determined, but malfunction of reproductive structures (19,54) and competition for metabolites (32) have been suggested. Senescing citrus flowers produce high levels of ethylene, but fruitlet abscission appears independent of ethylene production (111). Bloom drop may be intensified by diseases (35,36), insects (44) and temperature or water stress (23, 50).

A second peak of fruit drop, "June drop", occurs from mid-May to early July in California (22) and from late April to early June in Florida (19,46,78,125). Sweet orange losses during June drop range from insignificant (33) to almost total (122). Abscission typically occurs at the base of the ovary.

June drop is probably caused by competition between fruitlets for metabolites (32). High temperature, water stress, or both appear to accentuate the problem (23,56,122). Several fungi have been isolated from fruit during June drop, the most prevalent being Alternaria spp. (23,112,119). These fungi, however, also have been detected in healthy fruit, and attempts to control June drop with fungicides

have been unsuccessful (23,79,119). Fruit remaining on the tree after the June drop period typically reach commercial maturity with little further drop in most sweet oranges. The period from bloom until the end of June drop is considered the fruit-set period (67).

A third wave of drop in sweet oranges is preharvest drop. This drop affects fully developed fruit and becomes increasingly important during on-tree storage of fruit (5, 31,39,55). Some causes of preharvest drop are fruit splitting, brown rot (Phytophthora spp.), black rot (Alternaria citri Ellis & Pierce), stem-end rot (Diplodia natalensis Pole-Evans), twig dieback, and freeze injury (31, 55,62). Many apparently sound fruit, however, also fall for unknown reasons during prolonged storage on the tree (31).

Fruit drop continues after the fruit set period and before preharvest drop for navel orange in Florida (78,79). Summer drop, which occurs from early June until early August, and summer-fall drop,which occurs from late August through October, have been characterized (78,79). Summer and summer-fall fruit drop reduced yield by as much as 17% and 15%, respectively, in a navel grove in Florida in 1979 (79).

The major causes of navel fruit drop after fruit set are related to the morphology and anatomy of the fruit stylar-end and the secondary fruit. Summer drop is caused by a yellowing of the secondary fruit resulting from its abscission from the primary-fruit central axis (Chapter I, p. 41, and Chapter II, p. 63). Fruit splitting, stylar-end

decay, branch collapse and brown rot (Phytophthora spp.) are responsible for most of the summer-fall drop (79). Fruit splitting and stylar-end decay more frequently affect fruit with larger stylar-end aperture. In addition, most fruit affected by stylar-end decay have rind tissue protrusions from the secondary fruit into the affected locules (79). These protrusions are present in fruit locules before any decay has taken place and consist of secondary-carpel rind outgrowths (Chapter I, p. 38).

Control of Citrus Fruit Drop

Several attempts have been made to increase fruit set in citrus. Cross-pollination is recommended as a means of increasing fruit set and yield in Florida for 'Orlando' tangelo and other tangerine-type hybrids (67). Cross-pollination of navel orange with other citrus cultivars has been successful in South Africa (28) and Egypt (30), but not in Florida (67). Cross-pollination by bees is difficult because navel flowers produce little pollen and attract nectar-gathering but not pollen-gathering bees (28).

Girdling or scoring of the trunk or major branches during bloom generally reduces fruit drop during the fruit-set period and increases yield (66,67,71,86). Navel oranges, however, do not respond consistently to girdling. Yield increases were achieved in the first year of treatment in

some instances (66), but girdling over several successive years did not result in higher cumulative yield compared to untreated trees (6,107). Azzouni and Mahmoudi (9) reported girdling to be ineffective in increasing yield of navel orange in Egypt.

Some researchers have suggested excessive abscission of navel fruitlets to result from high temperature, wind, or water stress (23,50,67,73,122). Accordingly, overhead misting or application of an antitranspirant spray shortly after bloom resulted in higher leaf water potential and increased fruit set of navel orange in Florida (26). In addition, overhead sprinkling of navels in California during periods of high temperature significantly reduced air and leaf temperature and increased fruit set (15).

Hormones influence fruit set and development (40,61, 125). Applications of auxins (38,94,113) or cytokinins (38, 88,110), however, usually have little or inconsistent effects on citrus fruit set. Gibberellin (GA) applications to individual flowers, flower clusters or small branches consistently result in increased fruit set in navel orange as well as other citrus (38,48,68,109). Results for whole-tree GA applications, however, generally have been inconsistent. Hield et al. (48) obtained increased navel fruit set initially in California, but further attempts resulted in severe leaf drop. Similar attempts in Florida have failed to increase navel yield (68). Fruit set and yield of 'Orlando' tangelo and other tangerine hybrids,

however, usually are increased by whole-tree sprays of GA during bloom (21,69). More recently, spray applications of GA alone or in combination with $Ca(H_2PO_4)_2$ and/or 6-benzyladenine resulted in increased fruit set of navel orange in Florida (110). Yield data, however, were not obtained for these experiments. No material is presently recommended to improve fruit set of navel orange in Florida.

Auxin sprays can be used to control preharvest fruit drop of citrus in Florida (5,39) and California (31,62,113). Auxin action may be indirect through the control of fruit-stem dieback and water spot of navel orange (31,62), or direct by inhibition or delay of mature fruit abscission (5, 39,84).

Little information is available on the effects of growth regulator applications on summer and summer-fall fruit drop of navel orange in Florida. Sprays of 2,4-D at bloom or a few weeks later appeared to prevent summer drop (78), but information on summer-fall drop and yield was incomplete.

Materials and Methods

Plant Material

Fruit drop was studied from 1979 to 1982 in 2 navel orange (Citrus sinensis (L.) Osbeck)groves located near

Eustis in the north central citrus region of Florida. The rootstock in both groves was sour orange (Citrus aurantium L.). Grove A, planted in 1957 was used in 1979 and 1980 but later abandoned because of severe freeze injury. Grove B, planted in 1969, and unhurt by the 1981 and 1982 freezes, was chosen for studies in 1981 and 1982.

Experiments 1 and 2, 1980

Two experiments to control fruit drop were carried out in 1980. Each experiment consisted of 5 to 6 spray treatments with 5 single-tree replications arranged in a randomized complete block design. Treatments in experiment 1 were 20 ppm gibberellic acid (GA), 20 ppm 2,4-dichlorophenoxyacetic acid (2,4-D), 20 ppm 2,4-D + 20 ppm GA, 1000 ppm Promalin (Ciba-Geigy, 1.8% GA_{4+7} plus 1.8% N-phenylmethyl-1-H-purin-6-amine), and an unsprayed control. All treatments were applied on May 27, at 8 weeks after midbloom. Those of experiment 2 were 20 ppm 2,4-D at midbloom + 11 weeks (June 17), 20 ppm GA or 20 ppm 2,4-D at midbloom + 15 weeks (July 17), 20 ppm GA or 20 ppm 2,4-D at midbloom + 19 weeks (August 14), and an unsprayed control. Treatments were applied to run-off as sprays of 75 liters of solution per tree with a hand-gun sprayer. Midbloom occurred from March 25 to 31.

Experiment 3, 1981

Ten spray treatments for fruit drop control were evaluated in 1981 at grove B, using 4 single-tree replications in a randomized complete block design. Treatments consisted of 20 ppm GA, 10 or 20 ppm 2,4-D, and 10 or 20 ppm 2,4-D + 20 ppm GA applied at 3 dates, March 20 (midbloom), May 1 (midbloom + 5 weeks), and June 26 (midbloom + 13 weeks), and an unsprayed control. Treatments were applied to run-off as sprays of 75 liters of solution per tree with a hand-gun sprayer. Midbloom occurred from March 20 to 26.

Experiment 4, 1982

The 1982 experiment at grove B consisted of 5 treatments using 5 to 10 single-tree replications in a completely randomized design, trunk girdling at early bloom (February 19) or midbloom + 9 weeks (May 6), 20 ppm 2,4-D at midbloom + 6 weeks (April 19) or midbloom + 9 weeks (May 6), and an unsprayed, ungirdled control. Spray treatments were applied to run-off at 55 liters of solution per tree with a hand-gun sprayer. Trunk girdling consisted of a single knife-cut through the bark around the trunk at 10 to 20 cm above the bud union. Midbloom occurred from March 1 to 5.

Fruit Drop and Yield Evaluation, 1980 to 1982

Fruit drop was evaluated through catch-frame counts from bloom until the end of the fruit-set period in mid-June in 1981 (experiment 3), and, from mid-June until harvesting, by whole-tree counts in 1980 to 1982 (experiments 1 to 4). The catch-frame method consisted of placing a Saran cloth frame (2 x 1 m) tangentially to the canopy drip line in a similar location underneath each tree. Flower buds, flowers and fruit collected in the frames were counted and removed at 7- to 14-day intervals. The canopy projection area was estimated from diameter measurements. Total drop of reproductive structures per tree was calculated from the drop counts in the 2 m^2 catch-frames. Whole-tree fruit drop counts were made by counting fruit underneath each tree at 7- to 14-day intervals. An examination to determine cause of fruit drop was carried-out at this time.

Yield was determined in 1981 and 1982 by harvesting all remaining fruit on the trees shortly after commercial maturity in late November to early December. Number of boxes (40.8 kg each) per tree was obtained. Number of fruit per tree was calculated in 1981 from the number of fruit in each box, which in turn was estimated from fruit-diameter measurements made on 40 fruit per treatment (117).

Fruit External and Internal Characteristics, 1981 and 1982

The effects of spray treatments on fruit external and internal characteristics were evaluated in 1981 and 1982. Random samples of 10 fruit per tree were used for determination of external color, rind puncture force, juice content, total soluble solids (TSS), total (titratable) acid (TA), weight, equatorial diameter, stylar-end aperture diameter, peel thickness and secondary-fruit diameter.

External color was determined in a Hunter color difference meter using 2 readings per fruit taken at the fruit equator through a 5 cm-diameter viewing window. Lightness (L), redness (A) and yellowness (B) readings were recorded. The instrument was standardized using a white enamel plaque with values L = 94.0, A = -1.2, and B = 2.2. Fruit color was expressed as a ratio between the values in the green-to-red (A) and blue-to-yellow (B) scales.

Fruit rind puncture force was determined using a Chatillon DPP-30 strain gauge equipped with a cylindrical plunger beveled to a 1 x 6 mm rectangular surface at the tip. Two measurements per fruit were made at the fruit equator.

Juice extraction and analyses were performed as recommended by Wardowski et al. (121). Juice content was expressed as % by weight. Total soluble solids (TSS) were determined using a Brix hydrometer calibrated to read directly in % sucrose. Total (titratable) acid (TA) was

obtained by titrating a 25 ml aliquot of juice with 0.3125 N NaOH in the presence of phenolphthalein indicator, and expressed as % anhydrous citric acid.

Primary- and secondary-fruit diameters were measured at the equator of each fruit. Peel thickness was determined in the primary fruit at the stylar end as close to the secondary fruit as possible, and at the fruit equator.

Fruit weight, diameter, stylar-end aperture diameter, peel thickness and secondary-fruit diameter also were determined on healthy fruit and fruit affected by secondary-fruit yellowing, stylar-end decay, and splitting, using 30 to 60 randomly sampled fruit per category. The same characteristics were determined on similar samples of healthy fruit on the north bottom and south top canopy positions of 5 trees.

Mean Separation

Mean separation was done either by a t-test or a Duncan's multiple range test, utilizing Kramer's adaptation for experiments with a variable number of replications (65).

Results and Discussion

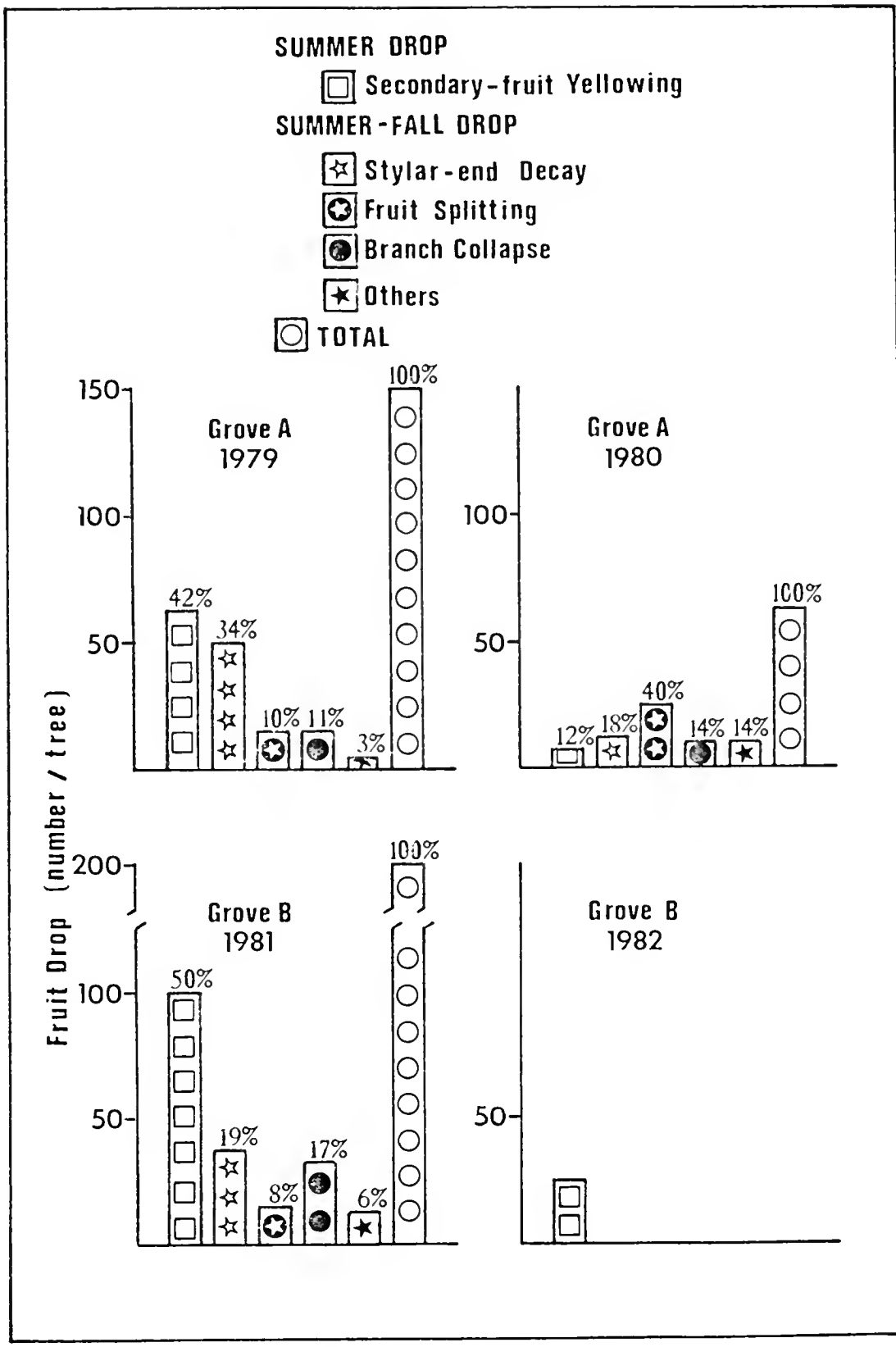
Summer and Summer-Fall Fruit Drop

Navel fruit drop after the fruit-set period was severe in both groves (Fig. 3.1). A total of 34% of the crop in 1981 was lost to fruit falling after the June drop, 15% to summer, 15% to summer-fall, and 4% to preharvest drop. Crop losses of 17% to summer drop and 15% to summer-fall drop had been reported previously (77).

Summer fruit drop. Summer drop was more severe in 1979 than 1980 in grove A, and in 1981 than 1982 in grove B (Fig. 3.1). Severe summer drop occurred in many navel orange groves throughout Florida in 1978, 1979 and 1981, while little summer drop occurred in 1980 and 1982, suggesting weather to be a major controlling factor. Years of heavy drop had a normal spring-flush and bloom, with midbloom occurring in the last week of March. Summer drop occurred shortly after the start of the summer flush. Conversely, 1980 and 1982 were years of an early and extended spring-flush and bloom with a less than normal summer flush, and little summer fruit drop.

Summer fruit drop occurred from early June until early August. Most fallen fruit were affected by secondary-fruit

Fig. 3.1. Distribution of causes of summer and summer-fall fruit drop in 2 navel orange groves near Eustis, Florida, 1979-1982. Summer-fall fruit drop was not determined in 1982. Causes of drop in the "others" category were brown rot (Phytophthora spp.) and undetermined causes. Bar superscripts indicate % in relation to total drop.


SUMMER DROP
Secondary-fruit Yellowing
SUMMER-FALL DROP
Stylar-end Decay
Fruit Splitting
Branch Collapse
Others
TOTAL
Fruit Drop (number / tree)
Grove A
1979
42%
34%
10%
11%
3%
100%
Grove A
1980
12%
18%
40%
14%
14%
100%
Grove B
1981
50%
19%
8%
17%
6%
100%
Grove B
1982
150
100
50
200

yellowing (SFY) which results from secondary-fruit abscission (Chapter II, p. 63).

Fruit affected by SFY had a larger stylar-end aperture (SEA) and a higher frequency of protrusions than healthy fruit, while other characteristics were similar (Table 3.1). Fruit with protrusions had larger secondary fruit and SEA diameter (Table 3.2). External or internal conditions leading to the development of larger SEA and higher frequency of protrusions may also result in higher incidence of secondary-fruit abscission and consequent SFY. Growth regulators affect the size of the SEA, e.g. flower dips with 2,4-D + GA solutions result in small SEA (68). Position of the fruit on the tree also influences the size of the SEA. Larger SEA have been associated with temperature and water stress of fruit located on the outside and upper portion of the tree (119). Leaf and fruit surface temperatures are higher and water potentials lower in the south top compared with the north bottom portion of the citrus tree canopy (115). Navel fruit in the south top were heavier and had larger secondary fruit, SEA, and a thicker peel than fruit in the lower portion of the canopy (Table 3.3).

Summer-fall fruit drop. Stylar-end decay (SED), fruit splitting, and branch collapse were major causes of summer-fall drop (Fig. 3.1), as previously reported (79). Summer-fall drop occurred from late August until late October and resulted in significant fruit losses in all seasons studied.

Table 3.1. Fruit weight, diameter, secondary-fruit diameter (SF), stylar-end aperture diameter (SEA), equatorial peel thickness and presence of secondary-fruit protrusions of healthy fruit and fruit affected by secondary-fruit yellowing (SFY) in navel orange, 1981.

Fruit characteristics[z]	Healthy	SFY-affected
Weight (g)	59	62ns
Diameter (mm)	50	51ns
SF diameter (mm)	11	11ns
SEA diameter (mm)	7	10**
Peel thickness (mm)	6	6ns
Protrusions (%)[y]	3	23

[z]Determined on June 17 on 30 randomly sampled fruit.

[y]Protrusions of SF rind tissue into primary-fruit locules, often associated with subsequent stylar-end decay.

ns,**Nonsignificant (ns) or significant at 1% level (**), by a t-test.

Table 3.2. Fruit weight, diameter, secondary-fruit diameter (SF), stylar-end aperture diameter (SEA), and equatorial peel thickness of healthy fruit and fruit containing rind protrusions (PRO) in navel orange, 1981.

Fruit characteristics[z]	Sampling date: June 17		Sampling date: September 26	
	Healthy	PRO[y]	Healthy	PRO[y]
Weight (g)	59	68ns	279	292ns
Diameter (mm)	50	53ns	82	84ns
SF diameter (mm)	11	14*	18	23**
SEA diameter (mm)	8	12*	14	18*
Peel thickness (mm)	6	6ns	4	4ns

[z]Determined on 30 randomly sampled fruit.

[y]Protrusions of SF rind tissue into primary-fruit locules, often associated with stylar-end decay.

ns,*,**Nonsignificant (ns) or significant at 5% (*) or 1% (**) level, by a t-test within each sampling date.

Table 3.3. Fruit weight, diameter, secondary-fruit diameter (SF), stylar-end aperture diameter (SEA), and equatorial peel thickness of fruit sampled at the north bottom and south top canopy positions in navel orange trees, 1981.

Fruit characteristics[z]	Canopy position	
	North bottom	South top
Weight (g)	310	369**
Diameter (mm)	84	90**
SF diameter (mm)	21	25**
SEA diameter (mm)	12	15*
Peel thickness (mm)	3.2	3.6*

[z]Determined on November 12, on 50 randomly sampled fruit.

*,**Significant at 5% (*) or 1% (**) level, by a t-test.

Stylar-end decay was characterized by decay around the secondary fruit typically restricted to 1 or 2 primary-fruit segments. The secondary fruit itself, however, was usually not affected. Fruit affected by SED were usually larger, had larger SEA, thicker peel, and higher frequency of secondary-fruit protrusions into primary-fruit locules, in 1980 and 1981 (Table 3.4). The locules which contained protrusions usually were the ones affected by SED. Previous studies had shown fruit with larger SEA were more affected by SED and the incidence of SED was related to the presence of secondary-fruit protrusions (77,79).

Fruit with large SEA and thinner peel at the stylar end are more affected by splitting (77). Fruit affected by splitting were typically heavier and had larger SEA in 1980 and 1981 (Table 3.5). In addition, split fruit had larger primary- and secondary-fruit diameters, and thicker peel in 1980 but not in 1981.

Control of Fruit Drop

Fruit set. Spray applications of 2,4-D, GA, or 2,4-D + GA at midbloom did not affect significantly bloom drop of navel orange in 1981 (Table 3.6). June drop, however, was accentuated by midbloom GA or 2,4-D + GA applications. This effect may have resulted from a delay in bloom drop since GA alone or with 2,4-D tended to reduce bloom drop (Table

Table 3.4. Fruit weight, diameter, secondary-fruit diameter (SF), stylar-end aperture diameter (SEA), equatorial peel thickness, and presence of rind protrusions of healthy fruit and fruit affected by stylar-end decay (SED) in navel orange, 1980 and 1981.

Fruit characteristics[z]	Sampling date			
	October 6, 1980		September 26, 1981	
	Healthy	SED	Healthy	SED
Weight (g)	—[x]	—	265	299**
Diameter (mm)	78	84*	81	85*
SF diameter (mm)	—	—	18	19ns
SEA diameter (mm)	7	16**	12	17*
Peel thickness (mm)	3.5	4*	4	5*
Protrusions (%)[y]	0	63	13	35

[z]Determined on 30 to 60 randomly sampled fruit.

[y]Protrusions of SF rind tissue into primary-fruit locules.

ns,*,**Nonsignificant (ns) or significant at 5% (*) or 1% (**) level, by a t-test within each sampling date.

[x]Not determined.

Table 3.5. Fruit weight, diameter, secondary-fruit diameter (SF), stylar-end aperture diameter (SEA), and equatorial peel thickness of healthy and split fruit in navel orange, 1980 and 1981.

Fruit characteristics[z]	Sampling date			
	October 6, 1980		September 26, 1981	
	Healthy	Split	Healthy	Split
Weight (g)	253	288**	265	280**
Diameter (mm)	78	82*	81	80ns
SF diameter (mm)	18	23*	18	17ns
SEA diameter (mm)	7	23**	12	20*
Peel thickness (mm)	3.5	4*	4	4ns

[z]Determined on 30 to 60 randomly sampled fruit.

ns,*,**Nonsignificant (ns) or significant at 5% (*) or 1% (**) level, by a t-test within each sampling date.

Table 3.6. Bloom and June drop of reproductive structures of navel orange as affected by gibberellic acid (GA) and 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Fruit drop (number per tree)[y]	
	Bloom drop[x]	June drop[w]
No spray	29,291a[v]	102b
Midbloom (March 20)		
GA	22,714a	761a
2,4-D	28,650a	279b
2,4-D + GA	25,538a	993a
Midbloom + 5 wks (May 1)		
GA	—[u]	111b
2,4-D	—	102b
2,4-D + GA	—	139b

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of solution per tree.

[y]Estimated from weekly counts in 1 catch-frame (2 m^2) per tree, and total canopy projection area; 4 single-tree replications per treatment.

[x]Drop of flower buds ca. 2 mm and larger in length and young fruit, from March 20 to April 25.

[w]Drop of young fruit from April 25 to June 19.

[v]Unlike letters within type of drop indicate significant differences by Duncan's multiple range test, 5% level. F value for bloom drop was significant at the 10% level.

[u]Not applicable.

3.6, F value significant at the 10% level). Earlier attempts to increase fruit set of navels in Florida with similar treatments were unsuccessful (68). More recently, however, GA sprays increased fruit set of navel orange in relation to unsprayed controls, but the difference diminished after June drop (110).

Sprays of 2,4-D, GA, or 2,4-D + GA at midbloom + 5 weeks had no effect on June drop of navelorange in 1981 (Table 3.6).

Summer fruit drop. A single spray of 10 or 20 ppm 2,4-D alone or in combination with 20 ppm GA at or within 9 weeks of midbloom resulted in significant control of summer fruit drop in 1980, 1981, and 1982 (Tables 3.7,3.8,3.9). Similar sprays at midbloom + 13 weeks in 1981 were less effective probably because summer drop had already begun by that time. Abscission of the secondary fruit precedes the development of SFY and subsequent summer fruit drop (Chapter II, p. 63). Application of 2,4-D to the fruit or fruit stem inhibited secondary-fruit abscission and SFY development (Chapter II, Table 2.7), which suggests a similar mechanisms for summer-drop control by whole-tree 2,4-D sprays.

An application of GA alone or Promalin at or within 13 weeks of midbloom did not influence summer fruit drop (Tables 3.7,3.8,3.9). Similarly, trunk girdling at early bloom or at midbloom + 9 weeks resulted in no change in summer fruit drop in 1982 (Table 3.9).

Table 3.7. Summer fruit drop of navel orange as affected by gibberellic acid (GA), 2,4-dichlorophenoxyacetic acid (2,4-D) and Promalin applications, 1980.

Spray treatments[z] (midbloom + 8 wks, May 27)	Summer fruit drop[y] (number per tree)
No spray	8a[x]
GA	7a
2,4-D	2b
2,4-D + GA	2b
Promalin	5ab

[z]Twenty ppm GA, 20 ppm 2,4-D or 1000 ppm Promalin were applied in 75 liters of spray solution per tree.

[y]Whole-tree counts made at 1- to 2-week intervals from June 20 to August 14; 5 single-tree replications per treatment.

[x]Unlike letters indicate significant differences by Duncan's multiple range test, 1% level.

Table 3.8. Summer fruit drop of navel orange as affected by gibberellic acid (GA) and 2,4-dichlorophenoxy-acetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Summer fruit drop[y] (number per tree)
No spray	101ab[x]
Midbloom (March 20)	
GA	123a
2,4-D	19c
2,4-D + GA	27c
Midbloom + 5 wks (May 1)	
GA	93ab
2,4-D	18c
2,4-D + GA	5c
Midbloom + 13 wks (June 26)	
GA	131a
2,4-D	59bc
2,4-D + GA	54bc

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Whole-tree counts made at 1- to 2-week intervals from June 19 to August 7; 4 single-tree replications per treatment.

[x]Unlike letters indicate significant differences by Duncan's multiple range test, 5% level.

Table 3.9. Summer fruit drop of navel orange as affected by trunk girdling or 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1982.

Treatments[z]	Summer fruit drop[y] (number per tree)
Control	26a[x]
Trunk girdling	
Early bloom (February 19)	21a
Midbloom + 9 wks (May 6)	25a
2,4-D sprays	
Midbloom + 6 wks (April 19)	3b
Midbloom + 9 wks (May 6)	4b

[z]Five single-tree replications per treatment. Girdling consisted of a single knife-cut through the bark around the trunk at 10 to 20 cm above the bud union. Applications of 2,4-D consisted of 20 ppm sprays of 55 liters of solution per tree.

[y]Whole-tree counts made at 1- to 2-week intervals from June 2 to August 6.

[x]Unlike letters indicate significant differences by Duncan's multiple range test, 1% level.

Summer-fall fruit drop. Growth regulator sprays at or within 19 weeks of midbloom generally had no effect on summer-fall fruit drop in 1980 (Table 3.10) and 1981 (Table 3.11) with 2 exceptions. A 2,4-D + GA spray at midbloom + 8 weeks in 1980 significantly reduced fruit drop (Table 3.10). This application did not specifically affect any of the causes of summer-fall drop (data not shown). Conversely, a 2,4-D + GA spray application at midbloom in 1981 increased summer-fall fruit drop (Table 3.11) by increasing the number of fruit affected by splitting (Table 3.12). Sprays of GA or 2,4-D at midbloom also increased fruit splitting (Table 3.12), but the resulting increase in summer-fall drop was not significant (Table 3.11). An increase in navel fruit splitting was reported following application of GA during bloom but not after later applications (49,70). None of the other causes of summer-fall drop were affected by spray applications (Table 3.12).

Preharvest fruit drop. Preharvest drop was not influenced by GA, 2,4-D or 2,4-D + GA sprays at or within 13 weeks of midbloom in 1981 (Appendix, Table A.1).

Yield. Growth regulators influenced fruit yield in 1981 (Table 3.13) but not in 1982 (Appendix, Table A.2). A 2,4-D spray at midbloom + 5 weeks, which had lessened summer drop (Table 3.8), also resulted in higher yield and number of fruit per tree in 1981 (Table 3.13).

Table 3.10. Summer-fall fruit drop of navel orange as affected by gibberellic acid (GA), 2,4-dichlorophenoxyacetic acid (2,4-D) and Promalin applications, 1980.

Spray treatments[z]	Summer-fall fruit drop[y] (number per tree)
Experiment 1	
No spray	55a[x]
Midbloom + 8 wks (May 27)	
GA	41ab
2,4-D	42ab
2,4-D + GA	32b
Promalin	47ab
Experiment 2	
No spray	47a
Midbloom + 11 wks (June 17)	
2,4-D	32a
Midbloom + 15 wks (July 17)	
GA	40a
2,4-D	35a
Midbloom + 19 wks (August 14)	
GA	48a
2,4-D	39a

[z]Twenty ppm GA, 20 ppm 2,4-D, or 1000 ppm Promalin were applied in 75 liters of spray solution per tree.

[y]Whole-tree counts made at 1- to 2-week intervals from August 14 to October 13; 5 single-tree replications per treatment.

[x]Unlike letters within experiments indicate significant differences by Duncan's multiple range test, 5% level.

Table 3.11. Summer-fall fruit drop of navel orange as affected by gibberellic acid (GA) and 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Summer-fall fruit drop[y] (number per tree)
No spray	99bc[x]
Midbloom (March 20)	
GA	145ab
2,4-D	144ab
2,4-D + GA	168a
Midbloom + 5 wks (May 1)	
GA	113bc
2,4-D	106bc
2,4-D + GA	102bc
Midbloom + 13 wks (June 26)	
GA	96c
2,4-D	107bc
2,4-D + GA	90c

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Whole-tree counts made at 1- to 2-week intervals from August 7 to October 29; 4 single-tree replications per treatment.

[x]Unlike letters indicate significant differences by Duncan's multiple range test, 5% level.

Table 3.12. Summer-fall fruit drop of navel orange due to stylar-end decay (SED), fruit splitting, branch collapse, and other causes, as affected by gibberellic acid (GA) and 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Summer-fall drop (number per tree)[y]			
	Causes of drop			
	SED	Splitting	Collapse	Other[x]
No spray	38a[w]	16c	33a	12a
Midbloom (March 20)				
GA	38a	46b	36a	25a
2,4-D	44a	47b	35a	18a
2,4-D + GA	31a	81a	39a	17a
Midbloom + 5 wks (May 1)				
GA	35a	19c	36a	23a
2,4-D	35a	20c	30a	21a
2,4-D + GA	34a	20c	32a	16a
Midbloom + 13 wks (June 26)				
GA	42a	18c	16a	20a
2,4-D	42a	21c	27a	17a
2,4-D + GA	41a	15c	18a	16a

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Whole-tree counts made at 1- to 2-week intervals from August 7 to October 29; 4 single-tree replications per treatment.

[x]Due to brown rot (*Phytophthora* spp.) and undetermined causes.

[w]Unlike letters within causes of drop indicate significant differences by Duncan's multiple range test, 5% level.

Table 3.13. Navel orange yield as affected by gibberellic acid (GA) and 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Yield per tree[y]	
	Boxes[x]	Number[w]
No spray	3.7c[v]	431b
Midbloom (March 20)		
GA	3.8bc	497ab
2,4-D	4.3abc	496ab
2,4-D + GA	3.8bc	497ab
Midbloom + 5 wks (May 1)		
GA	4.5abc	547ab
2,4-D	5.0a	584a
2,4-D + GA	4.5abc	570a
Midbloom + 13 wks (June 26)		
GA	4.8ab	524ab
2,4-D	4.3abc	498ab
2,4-D + GA	4.5abc	549ab

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Determined on December 5; 4 single-tree replications per treatment.

[x]Florida field box, equivalent to 40.8 kg of fruit.

[w]Estimated from number of fruit per box, which was calculated from fruit diameter (117).

[v]Unlike letters within columns indicate significant differences by Duncan's multiple range test, 5% level.

A combination of 2,4-D and GA at midbloom + 5 weeks also controlled summer drop (Table 3.8) and increased number of fruit per tree, but not yield, possibly because of a reduction in fruit size. Fruit from these 2,4-D + GA treatments weighed 321 g and had a diameter of 85 mm while corresponding values for control fruit were 335 g and 87 mm. A GA spray at midbloom + 13 weeks did not affect summer (Table 3.8) or summer-fall (Table 3.11) fruit drop in 1981, but resulted in higher yield (Table 3.13). This effect was possibly related to an increase in fruit size since the number of fruit per tree did not differ from that of unsprayed controls (Table 3.13). Fruit on trees given the GA treatment averaged 363 g and had a diameter of 89 mm while control fruit averaged 335 g and were 87 mm in diameter. Applications of GA at bloom had no effect (49) or reduced navel fruit size for 7 weeks following treatment (109). Applications of GA after petal fall, however, increased fruit size in navel and 'Pineapple' oranges, and 'Orlando' tangelo (70,88).

Applications of 2,4-D, GA, or 2,4-D + GA at midbloom, GA at midbloom + 5 weeks and 2,4-D or 2,4-D + GA at midbloom + 13 weeks had no effect on fruit number or yield (Table 3.13). Applications of 2,4-D, GA, or 2,4-D + GA at midbloom controlled summer drop (Table 3.8), but also resulted in increased fruit splitting (Table 3.12).

Spray applications of 2,4-D at 6 or 9 weeks after midbloom reduced summer drop in 1982 (Table 3.9), but had no

effect on yield (Appendix, Table A.2). Summer drop that year, however, was a minor problem, as only 26 fruit per tree were affected on unsprayed trees. Consequently, summer drop control by 2,4-D applications did not result in significantly higher yield.

Trunk girdling at early bloom or at midbloom + 8 weeks had no effect on summer fruit drop (Table 3.9) or fruit yield (Appendix, Table A.2).

Fruit External and Internal Characteristics

Spray applications of 2,4-D, GA, or their combination did not significantly affect fruit weight and diameter (Table 3.14). An application of GA at midbloom + 13 weeks, however, increased fruit weight but not diameter, when compared to GA or 2,4-D + GA applications at midbloom. Applications of GA during bloom reduced fruit size of 'Orlando' tangelo while postbloom applications increased it (70).

Applications of 2,4-D and other auxin-like growth regulators increase sweet orange fruit size, but the effect is dependent on time, concentration and cultivar (5,47,88). An increase in the fruit size of navel orange by 2,4-D sprays has been reported (100,113). Sprays of 12 to 24 ppm 2,4-D are effective on navel orange in California when applied within 3 months of petal fall (47). Ten to 20 ppm

Table 3.14. Fruit characteristics of navel orange as affected by gibberellic acid (GA) and 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Fruit characteristics[y] Weight (g)	Diameter (mm)	Secondary-fruit diameter (mm)
No spray	335ab[w]	87a	19b
Midbloom (March 20)			
GA	313b	85a	20b
2,4-D	345ab	88a	24ab
2,4-D + GA	315b	85a	19b
Midbloom + 5 wks (May 1)			
GA	325ab	86a	17b
2,4-D	341ab	87a	27a
2,4-D + GA	321ab	85a	23ab
Midbloom + 13 wks (June 26)			
GA	363a	89a	24ab
2,4-D	347ab	87a	24ab
2,4-D + GA	337ab	87a	22ab

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Determined on November 17, shortly after legal maturity, on 10 fruit per tree, 4 single-tree replications per treatment.

[x]Secondary-fruit rind outgrowths into primary-fruit locules.

[w]Unlike letters within columns indicate significant differences by Duncan's multiple range test, 5% level.

Table 3.14 — extended.

Stylar-end aperture diameter (mm)	Presence of protrusions (%)	Tertiary fruit (%)[x]	Peel thickness Stylar-end (mm)	Peel thickness Equator (mm)
13ab	5a	30ab	2.7ab	4.0a
12ab	10a	25ab	2.5bc	3.7ab
10b	5a	35ab	2.5bc	3.7ab
11b	5a	38ab	2.3c	3.5b
12ab	3a	18b	2.9a	4.0a
15a	8a	53a	2.8ab	4.0a
12ab	5a	48a	2.8ab	3.9ab
12ab	8a	45ab	2.8ab	3.9ab
15a	15a	38ab	2.5bc	3.5b
13ab	3a	40ab	2.6ab	3.7ab

2,4-D applications at or within 13 weeks of midbloom, however, had no influence on navel orange weight or diameter in 1981 (Table 3.14) or 1982 (Appendix, Table A.3) in this study. Small size is usually not a problem for navel orange in Florida.

Secondary-fruit diameter was increased by a 2,4-D spray 5 weeks after midbloom in 1981, but sprays at other times and with GA or 2,4-D + GA had no effect (Table 3.14). These materials had no effect on the diameter of the stylar-end aperture (SEA) in 1981 (Table 3.14) and 1982 (Appendix, Table A.3). Similarly, the % of secondary-fruit protrusions into primary-fruit locules was not influenced by growth regulator application in 1981 (Table 3.14). A 2,4-D spray 5 or 13 weeks after midbloom, however, resulted in larger SEA than a 2,4-D or 2,4-D + GA spray at midbloom. Flower dips with 2,4-D + GA solutions have been observed to stimulate growth of the stylar-end rind which completely covered the secondary fruit (68).

Applications of 2,4-D, GA, or 2,4-D + GA had no effect on the frequency of well-developed tertiary fruit at the secondary-fruit stylar-end (Table 3.14). Sprays of 2,4-D or 2,4-D + GA at midbloom + 5 weeks, however, did increase the frequency of tertiary fruit in relation to an application of GA alone. The 2,4-D spray also resulted in larger secondary fruit (Table 3.14). Sprays of 2,4-D at midbloom + 5 weeks may enhance development of the extranumerary fruit which are present in the stylar-end of navel oranges.

Peel thickness at the stylar-end and at the equator of the navel fruit was reduced by a 2,4-D + GA spray at midbloom in 1981 (Table 3.14). In addition, a 2,4-D spray at midbloom + 13 weeks reduced equatorial peel thickness. Sprays of 2,4-D prior to or during bloom generally increase peel thickness (100,113), but the effect of later applications is variable (20,47,100,113). Conversely, GA sprays at or shortly after bloom decreased peel thickness, while sprays during June drop increased it (49).

There was no effect of 2,4-D, GA, or 2,4-D + GA applications at or within 13 weeks of midbloom on external color of navel fruit at legal maturity in 1981 (Table 3.15 and Appendix, Table A.4) and 1982 (Appendix, Table A.6). A 2,4-D + GA spray at midbloom + 13 weeks, however, resulted in greener fruit than GA at midbloom, 2,4-D and 2,4-D + GA at midbloom + 5 weeks, or 2,4-D at midbloom + 13 weeks; however, no treatment produced commercially unacceptable green fruit. In addition, GA alone or in combination with 2,4-D at midbloom + 13 weeks resulted in increased fruit firmness compared with all other treatments (Table 3.15 and Appendix, Table A.5). Sprays of GA, especially in combination with 2,4-D during later stages of fruit development are known to delay color development and senescence, and increase firmness of citrus fruit rind (5, 20,84,89). No effect of 2,4-D sprays at or within 9 weeks of midbloom on rind puncture force was observed in 1982 (Appendix, Table A.6).

Table 3.15. Fruit external color and rind puncture force of navel orange as affected by gibberellic acid (GA) and 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Fruit characteristics[y]	
	Fruit color (A:B ratio)[x]	Rind puncture force (kg)[w]
No spray	-0.339ab[v]	2.8b
Midbloom (March 20)		
GA	-0.317a	2.8b
2,4-D	-0.348ab	2.7b
2,4-D + GA	-0.343ab	2.7b
Midbloom + 5 wks (May 1)		
GA	-0.336ab	2.8b
2,4-D	-0.294a	2.9b
2,4-D + GA	-0.303a	2.9b
Midbloom + 13 wks (June 26)		
GA	-0.345ab	3.2a
2,4-D	-0.309a	2.8b
2,4-D + GA	-0.392b	3.5a

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Determined at color break, on October 29. Two readings per fruit, 10 fruit per tree, 4 single-tree replications per treatment.

[x]Refers to the ratio between the values in the green-to-red (A) and blue-to-yellow (B) scales in a Hunter color difference meter. Lower A:B values for oranges at color break indicate a greener fruit.

[w]Determined with a Chatillon strain gauge equipped with a cylindrical plunger beveled to a 1 x 6 mm rectangular surface at the tip.

[v]Unlike letters within columns indicate significant differences by Duncan's multiple range test, 5% level.

There were generally no significant effects of 2,4-D, GA, or 2,4-D + GA sprays at or within 13 weeks of midbloom on juice quality in 1981 (Table 3.16 and Appendix, Tables A.7,A.8,A.9,A.10), and 1982 (Table 3.17). The only exception was a small reduction in juice content after a 2,4-D spray applied 6 weeks after midbloom in 1982 (Table 3.17). Treated fruit, however, met the requirements for legal maturity (117). A 2,4-D + GA spray at or 5 weeks after midbloom resulted in slightly lower juice content as compared to sprays of GA at midbloom + 5 or 13 weeks and 2,4-D sprays at midbloom + 13 weeks in 1981 (Table 3.16). In addition, the TSS:TA ratio of fruit on trees sprayed with 2,4-D + GA at midbloom + 5 weeks was higher than that of fruit similarly treated with GA alone. Other studies also indicate that 2,4-D and GA applications have little or no effect on juice characteristics of navel orange (21,49,113).

Summary

Fruit drop occurred in navel orange after the fruit-set period from 1979 to 1982 in Florida. Major causes of drop were secondary-fruit yellowing (SFY) during summer drop and stylar-end decay (SED), fruit splitting and branch collapse during summer-fall drop. Fruit affected by SFY, SED and splitting had larger stylar-end aperture than healthy

Table 3.16. Navel orange juice content, total soluble solids (TSS), total (titratable) acid (TA), and TSS:TA ratio as affected by gibberellic acid (GA) and 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Juice characteristics[y]			
	Content (% by wt)	TSS[x] (%)	TA[w] (%)	TSS:TA
No spray	56.7abc[v]	9.7a	0.70a	14.0ab
Midbloom (March 20)				
GA	56.5abc	9.6a	0.70a	13.8ab
2,4-D	56.3abc	9.8a	0.73a	13.6ab
2,4-D + GA	54.7c	9.6a	0.71a	13.5ab
Midbloom + 5 wks (May 1)				
GA	57.9a	9.4a	0.74a	12.8b
2,4-D	54.8bc	9.4a	0.68a	13.7ab
2,4-D + GA	54.4c	9.8a	0.66a	14.8a
Midbloom + 13 wks (June 26)				
GA	56.8ab	9.4a	0.68a	13.8ab
2,4-D	57.6ab	9.6a	0.71a	13.5ab
2,4-D + GA	56.0abc	9.4a	0.69a	13.7ab

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Determined at color break on October 29. Ten fruit per tree, 4 single-tree replications per treatment.

[x]Determined as sucrose with a Brix hydrometer.

[w]Determined as anhydrous citric acid by 0.3125 N NaOH titration.

[v]Unlike letters within columns indicate significant differences by Duncan's multiple range test, 5% level.

Table 3.17. Navel orange juice content, total soluble solids (TSS), total (titratable) acid (TA), and TSS:TA ratio as affected by 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1982.

Spray treatments[z]	Juice characteristics[y]			
	Content (% by wt)	TSS (%)	TA (%)	TSS:TA
No spray	51a[x]	9.6a	0.71a	13.6a
Midbloom + 6 wks (April 19)	48b	9.5a	0.67a	14.3a
Midbloom + 9 wks (May 6)	49ab	9.5a	0.72a	13.4a

[z]Twenty ppm 2,4-D in 55 liters of spray solution per tree.

[y]Determined at color break on October 27, on 10 fruit per tree, 5 single-tree replications per treatment. Total soluble solids were determined as sucrose with a Brix hydrometer, and TA as anhydrous citric acid by 0.3125 N NaOH titration.

[x]Unlike letters within columns indicate significant differences by Duncan's multiple range test, 5% level.

fruit. In addition, split and SED-affected fruit were larger and had thicker peel. Both SED- and SFY-affected fruit had a higher % of secondary-fruit protrusions into primary-fruit locules.

A spray application of 10 or 20 ppm 2,4-D alone or in combination with 20 ppm GA, but not of GA alone or Promalin, at or within 9 weeks of midbloom consistently controlled SFY and summer fruit drop in 1980 to 1982 seasons. Summer-fall and preharvest fruit drop generally were not affected by spray applications of 2,4-D, GA, 2,4-D + GA or Promalin at or within 19 weeks of midbloom.

Fruit weight, diameter, and stylar-end aperture diameter were not influenced by growth regulator sprays. A 2,4-D + GA spray at midbloom reduced peel thickness at the equator and at the stylar-end; whereas, 2,4-D at midbloom + 13 weeks reduced peel thickness at the equator only. Fruit juice quality was not affected by 2,4-D, GA, or 2,4-D + GA spray applications in 1981 and 1982, but juice content was slightly reduced by 2,4-D at 6 weeks after midbloom in 1982.

Although a spray application of 2,4-D alone or in combination with GA at or within 9 weeks of midbloom controlled summer drop and did not affect fruit quality, only the 2,4-D application at midbloom + 5 weeks in 1981 resulted in increased yield. A GA application at midbloom + 13 weeks in 1981 increased yield but not number of fruit

per tree, even though fruit drop was not affected. In addition, this treatment resulted in firmer peel without affecting other fruit characteristics.

APPENDIX

Table A.1. Preharvest fruit drop of navel orange as affected by gibberellic acid (GA) and 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Preharvest fruit drop (number per tree)[y]
No spray	21a[x]
Midbloom (March 20)	
GA	26a
2,4-D	28a
2,4-D + GA	24a
Midbloom + 5 wks (May 1)	
GA	20a
2,4-D	23a
2,4-D + GA	25a
Midbloom + 13 wks (June 26)	
GA	21a
2,4-D	23a
2,4-D + GA	21a

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Whole-tree counts made at 1- to 2-week intervals from October 29 to December 3; 4 single-tree replications per treatment.

[x]Unlike letters indicate significant differences by Duncan's multiple range test, 5% level.

Table A.2. Navel orange yield as affected by trunk girdling or 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1982.

Treatments[z]	Yield per tree[y] (boxes)
Control	7.8a[x]
Trunk girdling	
Early bloom (February 19)	7.7a
Midbloom + 9 wks (May 6)	6.8a
2,4-D sprays	
Midbloom + 6 wks (April 19)	8.0a
Midbloom + 9 wks (May 6)	7.4a

[z]Girdling consisted of a single knife-cut through the bark around the trunk at 10 to 20 cm above the bud union. Sprays consisted of 20 ppm 2,4-D in 55 liters of solution per tree. Five to 10 single-tree replications per treatment.

[y]Determined on November 27, using Florida field boxes, each equivalent to 40.8 kg of fruit.

[x]Unlike letters indicate significant differences by Duncan's multiple range test, 5% level.

Table A.3. Fruit weight, diameter, and stylar-end aperture diameter (SEA) of navel orange as affected by trunk girdling or 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1982.

Treatments[z]	Fruit characteristics[y]		
	Weight (g)	Diameter (mm)	SEA diameter (mm)
No treatment	317a[x]	85a	12a
Trunk girdling			
Early bloom (February 19)	—[w]	85a	12a
Midbloom + 9 wks (May 6)	—	87a	12a
2,4-D sprays			
Midbloom + 6 wks (April 19)	327a	86a	13a
Midbloom + 9 wks (May 6)	313a	84a	12a

[z]Girdling consisted of a single knife-cut through the bark around the trunk at 10 to 20 cm above the bud union. Sprays consisted of 20 ppm 2,4-D in 55 liters of solution per tree.

[y]Determined at color break, on October 27, on 10 fruit per tree, 5 single-tree replications per treatment.

[x]Unlike letters within columns indicate significant differences by Duncan's multiple range test, 5% level.

[w]Not determined.

Table A.4. Fruit external color of navel orange as affected by gibberellic acid (GA) and 2,4-dichlorophenoxy-acetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Fruit color (A:B ratio)[y]		
	Sampling dates		
	Oct. 20	Oct. 29	Nov. 17
No spray	-0.379a[x]	-0.339ab	0.057ab
Midbloom (March 20)			
GA	-0.396a	-0.317a	-0.080b
2,4-D	-0.424a	-0.348ab	0.024ab
2,4-D + GA	-0.422a	-0.343ab	0.038ab
Midbloom + 5 wks (May 1)			
GA	-0.404a	-0.336ab	0.008ab
2,4-D	-0.380a	-0.294a	0.072a
2,4-D + GA	-0.384a	-0.303a	0.107a
Midbloom + 13 wks (June 26)			
GA	-0.408a	-0.345ab	0.036ab
2,4-D	-0.404a	-0.309a	0.038ab
2,4-D + GA	-0.414a	-0.392b	-0.014ab

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Two readings per fruit, 10 fruit per tree, 4 single-tree replications per treatment and date, during color break. The ratio A:B refers to the values in the green-to-red (A) and blue-to-yellow (B) scales in a Hunter color difference meter. Lower A:B values for oranges at color break indicate a greener fruit.

[x]Unlike letters within dates indicate significant differences by Duncan's multiple range test, 5% level.

Table A.5. Fruit rind puncture force of navel orange as affected by gibberellic acid (GA) and 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Rind puncture force (kg)[y]		
	Sampling dates		
	Oct. 20	Oct. 29	Nov. 17
No spray	3.9b[x]	2.9b	2.8b
Midbloom (March 20)			
GA	4.2b	3.0b	2.7b
2,4-D	3.9b	3.0b	2.7b
2,4-D + GA	4.2b	3.0b	2.7b
Midbloom + 5 wks (May 1)			
GA	4.0b	2.9b	2.8b
2,4-D	3.9b	2.9b	2.9b
2,4-D + GA	3.8b	3.0b	2.9b
Midbloom + 13 wks (June 26)			
GA	4.7a	3.4a	3.2a
2,4-D	4.0b	3.0b	2.8b
2,4-D + GA	5.1a	3.6a	3.5a

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Two readings per fruit, 10 fruit per tree, 4 single-tree replications per treatment, during color break. Determined with a Chatillon strain gauge equipped with a cylindrical plunger beveled to a 1 x 6 mm rectangular surface at the tip.

[x]Unlike letters within dates indicate significant differences by Duncan's multiple range test, 5% level.

Table A.6. Fruit external color and rind puncture force of navel orange as affected by 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1982.

Spray treatments[z]	Fruit characteristics[y]	
	Fruit color (A:B ratio)	Puncture force (kg)
No spray	0.361a[x]	3.1a
Midbloom + 6 wks (April 19)	0.321a	3.1a
Midbloom + 9 wks (May 6)	0.326a	3.6a

[z]Twenty ppm 2,4-D in 55 liters of solution per tree.

[y]Determined at color break, on October 27; 2 readings per fruit, 10 fruit per tree, 5 single-tree replications per treatment. The ratio A:B refers to values in the green-to-red (A) and blue-to-yellow (B) scales in a Hunter color difference meter. Lower A:B values for oranges at color break indicate greener fruit. Puncture force was determined in a Chatillon strain gauge equipped with a cylindrical plunger beveled to a 1 x 6 rectangular surface at the tip.

[x]Unlike letters within columns indicate significant differences by Duncan's multiple range test, 5% level.

Table A.7. Fruit juice content of navel orange as affected by gibberellic acid (GA) and 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Juice content (% by wt)[y]		
	Sampling dates		
	Oct. 10	Oct. 20	Oct. 29
No spray	45.7bc[x]	52.4a	56.7abc
Midbloom (March 20)			
GA	46.4ab	53.8a	56.5abc
2,4-D	45.9bc	52.1a	56.3abc
2,4-D + GA	43.8c	51.9a	54.7c
Midbloom + 5 wks (May 1)			
GA	48.5a	53.8a	57.9a
2,4-D	45.5bc	50.3a	54.8bc
2,4-D + GA	45.3bc	50.5a	54.4c
Midbloom + 13 wks (June 26)			
GA	46.9ab	53.3a	56.8ab
2,4-D	46.7ab	53.3a	57.6ab
2,4-D + GA	44.6bc	53.0a	56.0abc

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Ten-fruit sample per tree, 4 single-tree replications per treatment, determined at color break.

[x]Unlike letters within dates indicate significant differences by Duncan's multiple range test, 5% level.

Table A.8. Total soluble solids in navel orange juice as affected by gibberellic acid (GA) and 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Total soluble solids (%)[y] Sampling dates Oct. 10	Oct. 20	Oct. 29
No spray	8.6ab[x]	9.1a	9.7a
Midbloom (March 20)			
GA	8.4abc	9.0a	9.6a
2,4-D	8.7a	8.9a	9.8a
2,4-D + GA	8.1c	8.7a	9.6a
Midbloom + 5 wks (May 1)			
GA	8.6ab	8.8a	9.4a
2,4-D	8.4abc	8.8a	9.4a
2,4-D + GA	8.5abc	8.8a	9.8a
Midbloom + 13 wks (June 26)			
GA	8.3abc	8.9a	9.4a
2,4-D	8.5abc	8.8a	9.6a
2,4-D + GA	8.2c	8.7a	9.4a

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Determined as sucrose with a Brix hydrometer, during color break. Minimum required for legal maturity until October 31, 9% (117). Samples of 10 fruit per tree, 4 single-tree replications per treatment.

[x]Unlike letters within columns indicate significant differences by Duncan's multiple range test, 5% level.

Table A.9. Total (titratable) acid in navel orange juice as affected by gibberellic acid (GA) and 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1981.

Spray treatments[z]	Total (titratable) acid (%)[y]		
	Sampling dates		
	Oct. 10	Oct. 20	Oct. 29
No spray	0.81a	0.72ab	0.70a
Midbloom (March 20)			
GA	0.75a	0.74ab	0.70a
2,4-D	0.83a	0.80a	0.73a
2,4-D + GA	0.80a	0.73ab	0.71a
Midbloom + 5 wks (May 1)			
GA	0.79a	0.77ab	0.74a
2,4-D	0.78a	0.71b	0.68a
2,4-D + GA	0.75a	0.73ab	0.66a
Midbloom + 13 wks (June 26)			
GA	0.80a	0.75ab	0.68a
2,4-D	0.82a	0.78ab	0.71a
2,4-D + GA	0.75a	0.74ab	0.69a

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Determined during color break as anhydrous citric acid by 0.3125 N NaOH titration, on samples of 10 fruit per tree, 4 single-tree replications per treatment.

[x]Unlike letters within dates indicate significant differences by Duncan's multiple range test, 5% level.

Table A.10. Ratio of total soluble solids (TSS) and total (titratable) acid (TA) in navel orange juice as affected by gibberellic acid (GA) and 2,4-dichlorophenoxyacetic acid (2,4-D) applications, 1981.

Spray treatments[z]	TSS:TA ratio[y]		
	Sampling dates		
	Oct. 10	Oct. 20	Oct. 29
No spray	10.7ab[x]	12.7a	14.0ab
Midbloom (March 20)			
GA	11.3a	12.2abc	13.8ab
2,4-D	10.5ab	11.2c	13.6ab
2,4-D + GA	10.2b	11.9abc	13.5ab
Midbloom + 5 wks (May 1)			
GA	10.9ab	11.5bc	12.8b
2,4-D	10.9ab	12.4ab	13.7ab
2,4-D + GA	11.3a	12.0abc	14.8a
Midbloom + 13 wks (June 26)			
GA	10.5ab	12.0abc	13.8ab
2,4-D	10.4ab	11.3bc	13.5ab
2,4-D + GA	11.0ab	11.8abc	13.7ab

[z]Twenty ppm GA and/or 20 ppm (except 10 ppm at midbloom) 2,4-D were applied in 75 liters of spray solution per tree.

[y]Determined during color break on samples of 10 fruit per tree, 4 single-tree replications per treatment. Total soluble solids were determined as sucrose with a Brix hydrometer, and TA as anhydrous citric acid by 0.3125 N NaOH titration.

[x]Unlike letters within dates indicate significant differences by Duncan's multiple range test, 5% level.

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I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

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