FLOWERING NEWSLETTER REVIEW

The making of virgin fruit: the molecular and genetic basis of parthenocarpy

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Abstract

Fruit set—the commitment of an angiosperm plant to develop fruit—is a key developmental process that normally occurs following successful fertilization. Parthenocarpy arises when fruit automatically develop in the absence of fertilization. This review uses parthenocarpic fruit development as a focal device through which to recapitulate and understand the molecular effectors that mediate and regulate fruit set. The review demonstrates that studies of parthenocarpy are providing vital insight into plant development, signaling and, potentially, high-value agricultural products.

Keywords: Auxin, fruit set, gibberellins, MADS-box, parthenocarpy, seedless fruit.

Overview

In flowering plants, two distinct but parallel fertilization events are ordinarily necessary for the development of the seed and any subsequent fruit tissues. Fertilization of the egg cell results in the development of an embryo, the progenitor of the successor sporophyte plant. The second male gamete fertilizes the heretofore diploid central cell of the female gametophyte, which leads to the development of the endosperm, a triploid, placenta-like nutritive tissue vital to the survival of the embryo (Box 1; Fig. 1A).

In most instances, this double fertilization is required for proper seed and fruit development (Dumas and Rogowsky, 2008), and early experiments in strawberry (Fragaria×ananassa) have shown that the removal of the fertilized seed suppressed further development of fruit (Nitsch, 1950). ‘Fruit set’ (Box 1) is the initiation of the developmental program that leads to the fruit; it depends on successful fertilization in most cases. Following fertilization, specific floral tissue(s) would enlarge and develop into structures which protect and facilitate dispersal of the fertile seeds (Sotelo-Silveira et al., 2014). These protective structures, fleshy or dry, are typically derived from the ovary wall leading to the ‘botanical fruit’ as in tomato, Annona (sugar apple), and Prunus persica (peach) (Box 1; Fig. 1B). Nevertheless, the fruit flesh in some plants is derived from non-ovary tissues, and hence the ‘accessory fruit’ such as strawberry and apple (Box 1; Fig. 1C).

Some plants, however, supersede the requirement for fertilization. This ability is known as ‘parthenocarpy’ (Box 1), which arises from Greek words for ‘virgin fruit’. Parthenocarpy has contributed to perhaps some of humanity’s oldest domestic crops: it appears to have figured prominently in the domestication of breadfruit, banana, and fig (Fig. 2A, B) (Zerega et al., 2004; Kislev et al., 2006; Sardos et al., 2016). That some plants may produce fruit without the need for fertilization by the male gamete raises an important question of basic science—how do such plants trigger a profound development
program without the biochemical contribution normally required from sperm? It also raises critical technological questions, as parthenocarpy supplies several direct and practical benefits.

Progress has been made across several plant species toward an understanding of the mysteries of virgin fruit. Classic scientific experiments demonstrated that phytohormones can induce parthenocarpy; more recent research has uncovered the molecular mechanisms through which these phytohormones achieved such effects. Table 1 provides a summary list of genes and phytohormones previously linked to parthenocarpy. The bulk of the review will focus on the progress made toward identifying gene products that guide fruit development—genes that act as producers and perceivers of signals—and how changes to these signals and signal sensors have led to parthenocarpy. This review argues that recent genetic advances in model systems, particularly in Arabidopsis thaliana and tomato, have left the field well positioned for much deeper research into plants of greater agricultural significance. That transition has begun; much work remains before us.
This review will not, however, seek to encompass some subjects closely related to parthenocarpy. It will not cover apomixis (Box 1), which is also asexual reproduction, but which results in the development of an embryo from unreduced female tissue. The review also will not cover stenocarpicarpy (Box 1)—seedlessness or near seedlessness—caused by the spontaneous or induced abortion of a fertilized seed; this process's dependence on fertilization distinguishes it from parthenocarpy, which has no requirement for sperm or fertilization at any point.

Indeed, the distinction between parthenocarpy and stenocarpocarpy underscores some of the key benefits of parthenocarpy (Ruan et al., 2012). First, pollination can be costly, can require specific pollinators, and generally demands specific temperature and other ambient conditions. All of these requirements are threatened by climate change, disease, or other phenomena. Secondly, parthenocarpic fruit is seedless fruit, which is preferred by many consumers, and requires less processing in many agricultural applications. Thirdly, parthenocarpy results in precocious development of the endosperm, which is the principal source of nutritional and agricultural value in many plants. Finally, parthenocarpy provides insight into the mechanisms governing endosperm development and fruit set.

Because of its diverse origins, studies of parthenocarpy have encompassed a wide range of subjects in plant signaling and development, and continued study also should allow practically oriented, fundamental research science. This review begins with discussion of phytohormones in fertilization-induced fruit development, highlights emerging insights into epigenetic regulation of fertilization, and underscores the importance of MADS-box transcription factors. It concludes with final thoughts on major opportunities lying ahead for this exciting field of research.

The role of hormones in parthenocarpy

The earliest observations of parthenocarpy involved natural mutations. Insight into molecular explanations of parthenocarpy began with the study of the role of phytohormones. Auxin was the phytohormone first identified as capable of inducing parthenocarpic fruit development, in citrus and later in strawberry (Gustafson, 1939; Nitsch, 1950). These experiments showed that ovaries generate auxin signals that induce development of both botanical and accessory fruits, and that ectopic supply of auxin-like substances would have similar effects. Gibberellin acid (GA) later was shown to have a similar effect on roses and apples (Prosser and Jackson, 1959; Davison, 1960). Much later, ectopic cytokinins were shown to induce parthenocarpy in watermelon and kiwifruit (Hayata and Niimi, 1995). In contrast to the potential of auxins or GA to induce parthenocarpy, abscisic acid (ABA) and ethylene signals have not been shown to be positively related to parthenocarpy. Indeed, ABA levels are low in the ovaries of parthenocarpic mandarin oranges (Mesejo et al., 2010), and ethylene has been shown to suppress fruit set in tomato (Shinozaki et al., 2015).

These early insights into the role of phytohormones led to a wave of discoveries related to the pathways necessary to metabolize and sense these hormones. Focusing particularly on auxin and GA, where the bulk of research has been done, parthenocarpic mutations were found in a variety of species. Research into cytokinin-related parthenocarpy is less advanced at present.

Auxin-related parthenocarpy has been found in a number of steps within the auxin synthesis, perception, and signaling pathways. Transgenic eggplants (Solanum melongena) and tobacco showed enlarged and parthenocarpic fruits due to the ovule-specific expression of an auxin biosynthesis gene DefH9::iaaM (Rotino et al., 1997). Overexpression of the auxin receptor TIR1 in tomato gives a parthenocarpic phenotype (Ren et al., 2011). Similarly, the suppression of auxin signal repressors is associated with parthenocarpic. For instance, a loss or reduction of function in the tomato IAA9 gene resulted in parthenocarpic seedless tomato fruit (Wang et al., 2005; Mazzucato et al., 2015), and in eggplant, RNA-seq data also link reduced expression of indole acetic acid (IAA) repressor genes with parthenocarpic fruit development (Chen et al., 2017). Similarly, removing the function of negative regulators of auxin signaling encoded by AUXIN RESPONSE FACTOR 8 (AtARF8) in A. thaliana and tomato and ARF7 in tomato also led to fertilization-independent fruit development (Goetz et al., 2006, 2007; de Jong et al., 2009b) (Fig. 2C–F). In another example, RNAi silencing of the AUCSIA genes encoding plant peptides that restrain auxin response was shown to lead to parthenocarpic fruit development (Molesini et al., 2009). Together, these experiments reinforce classic auxin experiments, providing molecular-level evidence that auxin biosynthesis and signaling regulate fruit set and that constitutive auxin signaling could lead to parthenocarpic fruit. Auxin appears to be an essential signal that relieves the typical repression of a fruit set program.

Changes in the GA signaling pathways also have been shown to induce parthenocarpic fruit set and fruit development. After the early findings that ectopic application of GA to rose and apple flowers and ovaries led to fruit set, later experiments in manually deseeded or emasculated strawberries showed that the application of GA could still induce enlargement of the fruit-like receptacle tissue, even in the absence of pollination (Thompson, 1969; Kang et al., 2013). In the heavily studied pat varieties of parthenocarpic tomatoes, aberrant expression of the GA pathway is a consistent characteristic (de Jong et al., 2009a), with GA3P, the GA precursor, accumulating at higher levels in unfertilized ovaries than in wild-type counterparts (Fos et al., 2000; Olimpieri et al., 2007). Overexpression and ectopic expression of gibberellin 20-oxidase—the enzyme that completes the final metabolic step in production of bioactive GA—leads to production of parthenocarpic fruit in tomato and in Arabidopsis (García-Hurtado et al., 2012). The key repressor of GA signal transduction is the DELLa protein, and, concomitantly, reduction in DELLA activity has been shown to lead to constitutive GA signaling phenotypes that have included parthenocarpic fruit development. For instance, reduced DELLA activity in the proceras loss-of-function mutation or via RNAi
Fig. 2. Photos of parthenocarpic fruits in comparison with fertilized fruits. (A) Fruits of wild and seeded banana (Musa acuminata banksia). (B) Domesticated parthenocarpic banana fruit. (C) A fertilization-induced tomato fruit of the Monalbo variety. (D) The ‘Monalbo’ wild-type fruit cut in half. The locule is filled with pulp and the seed are clearly visible. (E) A parthenocarpic fruit from ‘Monalbo’ containing the abnormal arf8 mutant transgene. (F) The same parthenocarpic fruit in (E) cut in half. The central columella (c) is enlarged and the locule is filled with pulp, showing a seedless endocarp. Scale bars=1 cm in (C–F). Images (A) and (B) are from Sardos et al. (2016) under Creative Commons Attribution License; images in (C)–(F) are from Goetz M, Hooper LC, Johnson SD, Rodrigues JC, Vivian-Smith A, Koltunow AM. 2007. Expression of aberrant forms of AUXIN RESPONSE FACTOR8 stimulates parthenocarpy in Arabidopsis and tomato. Plant Physiology 145, 351–366, with permission (Copyright American Society of Plant Biologists).

Table 1. Genes and phytohormones involved in parthenocarpic fruit development

| Target                        | Cause/treatment                      | Underlying pathway | Species                      | Source                                                                 |
|-------------------------------|--------------------------------------|--------------------|------------------------------|------------------------------------------------------------------------|
| Auxin (naphthoxyacetic acid)  | Exogenous application                | Auxin              | Strawberry (Fragaria ananassa and F. vesca) | Nitsch (1950); Kang et al. (2013)                                      |
| YUCCA                         | Overexpression                       | Auxin              | Eriobotrya japonica          | Mesejo et al. (2010)                                                   |
| ARF-7/8                        | Underexpression                      | Auxin              | A. thaliana; S. lycopersicum | Goetz et al. (2007); de Jong et al. (2008)                             |
| IAA                           | Differential expression found in natural parthenocarpic mutant | Auxin              | S. melongena                 | Chen et al. (2017)                                                     |
| AUCSI A                       | RNAi                                | Auxin              | S. lycopersicum              | Molesini et al. (2009)                                                 |
| PIN-4                         | RNAi                                | Auxin              | S. lycopersicum              | (Mouret et al., 2012)                                                  |
| Gibberellic acid              | Exogenous application                | GA                 | R. rugosa; M. domestica; F. vasa | Davison (1960); Prosser and Jackson (1959); Kang et al. (2013)        |
| GA20OX                        | Overexpression                       | GA                 | A. thaliana; S. lycopersicum | Garcia-Hurtado et al. (2012)                                           |
| DELLA                         | RNAi; loss-of-function mutations     | GA                 | A. thaliana; S. lycopersicum | Marti et al. (2007); Fuentes et al. (2012)                             |
| Cytokinin                     | Exogenous application                | Cytokinin          | C. lanatus; A. delicosa      | Hayata and Nirim (1995); Lewis et al. (1996)                           |
| MET1                          | RNAi                                | DNA methylation    | A. thaliana                  | FitzGerald et al. (2008); Schmidt et al. (2013)                        |
| MEDEA                         | Loss-of-function mutation            | PRC2 histone       | A. thaliana                  | Köhler et al. (2003)                                                   |
| FIS2                          | Loss-of-function mutation            | PRC2 histone       | A. thaliana                  | Chaudhury et al. (1997)                                                |
| FIE                           | Loss-of-function mutation            | PRC2 histone       | A. thaliana                  | Ohad et al. (1996)                                                     |
| MSI                           | Loss-of-function mutation            | PRC2 histone       | A. thaliana                  | Guitton and Berger (2005)                                              |
| PISTILLATA                    | Gain- and loss-of-function mutations | MADS-box           | Malus domesticus; Vitis vinifera | Yao et al. (2001); Fernandez et al. (2013)                             |
| DEFCIENS                      | Loss-of-function mutation            | MADS-box           | Elaeis guineensis            | Ong-Abdullah et al. (2015)                                             |
| SEP1/TM29                     | Antisense or co-suppression          | MADS-box           | S. lycopersicum              | Ampomah-Dwamena et al. (2002)                                          |
has led to unfertilized fruit set in tomato and silique development in Arabidopsis (Martí et al., 2007; Bassel et al., 2008; Fuentes et al., 2012). Parthenocarpy in the Arabidopsis della mutant led to siliques that are shorter than normal; the addition of auxin corrects this deficiency. However, this corrective effect of auxin was lost when GA synthesis was blocked by mutations, suggesting that the auxin input is upstream of GA biosynthesis (Fuentes et al., 2012). This crosstalk between auxin and GA signaling pathways has also been found in tomato (Serrani et al., 2008; Ding et al., 2013).

Cytokinins also have induced parthenocarpic fruit development in species including watermelon (Citrullus lanatus), Japanese pears (Pyrus pyrifolia), and kiwifruit (Actinidia delicosa) (Hayata and Niimi, 1995; Lewis et al., 1996; Kadota and Niimi, 2003). Cytokinin’s two-component signal transduction pathway is quite different from that of auxin or GA. Instead, the signal is relayed by a series of phosphorylation events transferred to effector proteins and eventually genes (Stock et al., 2000). As such, constitutively active cytokinin signal transduction components—rather than knockouts of negative regulators—are the most likely genetic source of parthenocarpy. To date, however, very little research has been conducted on such plants with such mutations; this may represent an important research opportunity.

Hormone transport should play a key role in fruit set, as the phytohormone-producing tissue, the seed, is spatially situated next to, on top, or beneath the phytohormone-sensing fruit tissues. RNAi knockdown of an ovary-specific auxin efflux transport protein has been shown to lead to production of parthenocarpic fruit in tomato (Mounet et al., 2012), perhaps reflecting an accumulation of excess auxin in the ovary. Transcriptome-level research in strawberry and tissue-specific expression of a dominant negative IAA allele in Arabidopsis show that auxin generated in the endosperm within a seed initiates fruit set and the effect of auxin on fruit set relies on proper transport from the site of synthesis to the target fruit tissue (Kang et al., 2013; Figueiredo et al., 2015). These studies highlight the endosperm within the seed as the source of auxin.

To summarize, a number of phytohormones have been shown to induce parthenocarpic fruit development in a number of plant species. Similarly, changes in the synthesis and metabolism of hormones or components within the signaling pathway may also induce fruit development in the absence of fertilization. Very broadly, this research emphasizes that changes to a plant’s ability to generate and perceive the ‘fruit set program’ signal are central to potential parthenocarpy.

Epigenetic mechanisms in fruit set

The critical role of epigenetic mechanisms in regulating complex development programs and tissue differentiation has long been appreciated (Allis and Jenuwein, 2016), and the importance of such mechanisms is arguably elevated further in plants than in motile animals. As such, it is not surprising that epigenetic mechanisms appear to play a key role in fruit development. Epigenetic regulation occurs at the transcriptional and post-transcriptional time points and at three distinct levels: at chromatin, DNA, and mRNA. This review argues that regulation or disturbance at each level can contribute to fertilized or parthenocarpic fruit set, respectively.

Two major mechanisms of transcriptional regulation are via histone modifications in chromatin and the methylation of cytosine in the underlying DNA strands. In seeds and fruit, the major mechanism responsible for histone modification is the Polycomb Repressive Complex 2 (PRC2), and the predominant mechanism regulating DNA methylation is RNA-directed DNA methylation (Gehring and Satyaki, 2017). DNA methylation, in which a methyl group is attached to the fifth carbon of cytosine residues within a DNA transcript, generally leads to reduced expression of a gene. In Arabidopsis, DRM2 acts as the major de novo methyltransferase enzyme and is largely regulated by RNA-directed DNA methylation (Stroud et al., 2012; Borges and Martienssen, 2015). MET1 and CMT3 are the primary maintenance methyltransferases, responsible for reproducing DNA methylation during DNA replication (Bartee et al., 2001; Kankel et al., 2003). Together, these systems of methylation provide exquisite, gene-level transcriptional control, and are critical to establishing the fates of differentiated tissues.

Operating at the genomic level, PRC2, which is conserved across fungi, animals, and plants (Lewis, 2017), adds repressive modifications to amino acid residues on the ‘tails’ of nucleosome histones. Products of several genes together comprise the Arabidopsis PRC2; most prominent in the fate of the seed are a group that contribute to the ‘FIS’, or Fertilization Independent Seed, version of the complex (Hands et al., 2016). These genes consist of MEDEA (MEA), a homolog of the Drosophila melanogaster gene Enhancer of Zeste; FERTILIZATION INDEPENDENT SEED 2 (FIS2), a homolog of the Drosophila gene Suppressor of Zeste; FERTILIZATION INDEPENDENT ENDOSPERM (FIE), homologous to the Drosophila Extra sex combs; and MULTICOPY SUPPRESSOR OF IRA1 (MSI1), homologous to p55 in Drosophila (Ohad et al., 1996; Chaudhury et al., 1997; Köhler et al., 2003; Guitton and Berger, 2005).

Perturbation in DNA methylation and the function of PRC2 have been shown to contribute toward parthenocarpic phenotypes. met1 plants have precocious and overproliferative integuments surrounding the female gamete (FitzGerald et al., 2008). Arabidopsis mutants defective in the PRC2-component genes have been linked to fertilization-independent seed development and precocious development of the silique fruit tissues in Arabidopsis (Chaudhury et al., 1997; Goodrich et al., 1997). This fertilization-independent seed development, however, does not lead to a fully viable seed but rather to a relatively early abortion of seed development mediated by a mechanism of programmed cell death (Chaudhury et al., 1997). Arabidopsis heterozygous for a knock down allele of Met1 and MEA are substantially more likely to cause fertilization-independent endosperm development than the mea-only heterozygote (Schmidt et al., 2013), demonstrating linkage between the two epigenetic pathways and reinforcing the role of DNA methylation in preserving the
seed ‘state’ (unfortunately, the study did not indicate whether *mel1mea* mutants also demonstrated the fertilization-independent silique elongation phenotype). Nonetheless, despite the pronounced phenotype found in Arabidopsis, inactivation of PRC2 component homologs in other plants does not have demonstrated parthenocarpic effects. This may be due to redundancy in these genes in other species. Nevertheless, research on FIS-type genes in other plants remains active (Luo et al., 2009; Nallamilli et al., 2013; Boureau et al., 2016; Liu et al., 2016).

In Arabidopsis, it was found that the PRC2 complex represses AGAMOUS-LIKE 62 (AGL62), which encodes a MADS-box transcription factor and acts to prevent premature cellularization in the endosperm tissue (Kang et al., 2008). Upon fertilization, AGL62 is expressed in the Arabidopsis endosperm and promotes the transport of auxin to the seed coat for proper seed coat development (Figueiredo et al., 2015, 2016). Loss of the PRC2 complex allows the continued expression of AGL62 in the central cell, thereby leading to automatic endosperm development (Kang et al., 2008). Although much remains unknown regarding the mechanism of epigenetic regulation during seed set and fruit set, fertilization-induced de-repression may underlie the fertilization-induced signal production and transduction for both seed set and fruit set.

**Certain MADS-box genes encode repressors of fruit development**

It has been appreciated for many years that, early in the flower development process, mutations in the gene PISTILLATA (*PI*), a MADS-box transcription factor and a B class gene, controls petal and stamen floral organ identity in Arabidopsis (Goto and Meyerowitz, 1994). This homeotic gene, however, appears to have broader functions in other species. In apple (*Malus domestica*), two cultivars sharing similar retrotransposon-mediated splicing variants of the *AtPI* homolog produced parthenocarpic fruit as well as mutant floral morphology that closely resembled that of the Arabidopsis *pi* loss-of-function mutants (Yao et al., 2001). Constitutive overexpression—also conferred by transposon insertion—of a *PI* homolog in grape (*Vitis vinifera*) blocked fruit set despite pollination (Fernandez et al., 2013). DEFICIENS (*DEF*), like *PI*, is a B class MADS-box gene regulating petal/stamen identity in snapdragon (Sommer et al., 1990). In oil palm (*Elaeis guineensis*), hypomethylation of a retrotransposon leads to alternative splicing of the oil palm homolog to *DEF*, which in turn produces aberrant floral structures and parthenocarpic fruit development (Ong-Abdullah et al., 2015).

In tomato, deficiencies in expression of *stamenless*, a *DEF* homolog, lead to development of ovule tissue and occasional parthenocarpy (Mazzucato et al., 2008). The E-class floral homeotic genes also encode MADS-box proteins, which form complexes with B and C class genes (Pelaz et al., 2000; Honma and Goto, 2001). Consequently, reduced expression in the tomato E-class gene *TM29* also led to parthenocarpic fruit development reminiscent of the B-class mutants (Ampomah-Dwamena et al., 2002). Together, these results suggest that B- and E-class MADS-box proteins may play a negative regulatory role in fleshy fruit development. When it is inactivated, fruit will automatically develop in the absence of fertilization. The study also pointed to a potential link between floral organ identity and fruit development.

Other MADS genes also regulate parthenocarpy. Recently, a parthenocarpic tomato mutant was identified in a clever mutagenesis screen for fruit-bearing tomato plants under extreme heat conditions that ensured pollen were no longer fertile. The mutant was found to be defective in the *SIAGL6* gene, and CRISPR/Cas9 (clustered regularly interspaced short palindromic repeats/CRISPR-associated protein 9) gene knock down of *SIAGL6* confirmed the phenotype (Klap et al., 2017). The work on B- and E-class floral homeotic genes and *AGL6* indicates that certain MADS-box genes encode repressors of fruit development.

**Conclusions and future directions**

This review summarized several molecular mechanisms involved in parthenocarpic fruit set. Following early agricultural observations and scientific results, it has been shown that fruit set is a response to fertilization-induced phytohormone surge and transport. That changes to the synthesis and degradation of such hormones or perturbations of a plant’s ability to perceive such hormonal signals might also induce parthenocarpic fruit development is a logical outgrowth of those initial findings. As scientific understanding of the mechanisms has grown, significant progress has been made in understanding the mechanisms of parthenocarpy. Many such alterations have been uncovered across a wide variety of model and non-model systems. The relationship between auxin signaling and parthenocarpic fruit set was uncovered first, and knowledge of parthenocarpic-inducing modifications to this pathway are most advanced; opportunities remain to exploit the relationship of GA and especially cytokinin signaling pathways to parthenocarpy.

Epigenetics provides governing mechanisms for fertilization-mediated release of phytohormone signals. DNA methylation, reinforced by PRC2-mediated histone alterations, has been shown to contribute to the activation of endosperm and fruit development, and alterations in this regulatory relationship lead to fertilization-independent development. MADS-box genes appear to be a key link between floral development, fruit set, and parthenocarpy. While past research has uncovered these multiple, complex factors, we know from the earliest studies of angiosperms that fertilization is the central signal. It is therefore very possible, and indeed logical, to postulate that endosperm development and fruit set are linked to the same ultimate fertilization signal or master regulator, although the molecular basis of that cascade of signals remains to be discovered.

Research into parthenocarpy is directly relevant to a number of pressing agricultural concerns. It also provides a precise lens into the molecular mechanism of fertilization—a process incompletely understood in both plant and animal
systems. The key messages of this review are that substantial progress has been made in answering the elegant, elusive questions surrounding a fruit’s virgin birth, and that significant opportunities remain for future work. Scientists, equipped with a significant knowledge base and new technology such as the CRISPR/CAS9 technology, are poised to improve agricultural productivity through parthenocarpy. Scientific prospects of this field are rather exciting.

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