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Role of plant hormones in flowering and exogenous hormone application in fruit/nut trees: a review of pecans

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Abstract

Pecan is the only native north American tree nut. The USA produces approximately 80% of the world's pecans. Pecan trees have an extended juvenility, 10 years to the first nut crop. With mature bearing they begin alternate bearing; alternating large and small crops. Theoretically, a heavy crop inhibits flower induction in the current year resulting in a low crop the following year. The flowering of perennial trees involves a complex interplay of multiple hormones. The possible molecular mechanisms regulating tree flowering can be revealed by endogenous plant hormone quantification, exogenous hormone application and RNA-sequencing. In this review, we synthesize the investigations of transcriptomic analysis and exogenous hormone treatments on bud break and flowering in fruit/nut trees with a focus on pecan. Knowledge of how hormones regulate flowering suggest they are a potential tool for improving return bloom and mitigating alternate bearing.

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Introduction

Pecan nuts (*Carya illinoensis*) are commercially valuable for both their medicinal and nutritional properties. Pecan nuts contain lipids (primary oils), carbohydrates, proteins, calcium (Ca), phosphorus (P), magnesium (Mg), vitamins A, B, and E, and volatile compounds[1]. Pecan, native to North America is grown in over 20 countries including Australia, Argentina, Mexico, Italy, France, Japan, and China[2]. The United States is the world's major producer supplying 80% of the global market[3]. Pecan is a monoecious tree that produces male (staminate or catkins) and female (pistillate) inflorescences on different parts of the same tree[4] (Fig. 1). The catkins are produced from primary compound buds of one-year-old branches, whereas pistillate flowers are developed from the terminal bud of the current season's shoot. The female inflorescence is a star-shaped terminal raceme (consisting of 4–5 flowers)[5]. Pecan produces a large number of staminate to pistillate flowers per branch. Pecan is characterized by alternate bearing (AB), alternating large and small crops precipitated by the lack of floral induction on heavy crop years, resulting in decreased flowering and yield the following year. During the transition of the primordium from a vegetative to fruit bud, including floral induction, initiation, and differentiation, the transition is controlled by environmental conditions and hormones, the latter generated by the plant in response to its carbohydrate status[6–8]. Plant hormone signaling, tree vigor, carbohydrate status, nutritional profile, and homeostasis are important factors that regulate bud and flower formation and development.

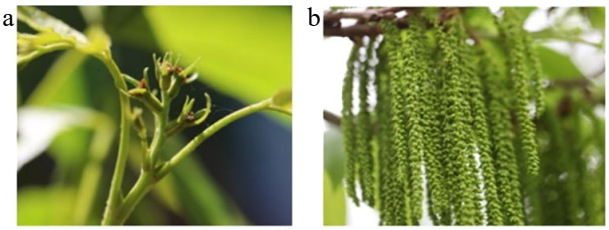

Fig. 1 (a) Pecan pistillate flowers; (b) pecan staminate flowers.

Auxin, cytokinins (CTK), gibberellins (GA), ethylene, and abscisic acid (ABA) are the major plant hormones. Jasmonic acid (JA), salicylic acid (SA), brassinosteroid (BR) are additional phytohormones thought to be involved in numerous plant physiological processes. Auxin is a key component in the development of flower primordia, without auxins the plant cannot form flowers[9]. The CTKs are involved in the regulation of growth and differentiation, including cell division, apical dominance, nutrient metabolism, chloroplast development, senescence, flowering, nodulation, and circadian rhythms[10,11]. One of CTK's major roles in flowering is delaying senescence. They also play a role in cell differentiation in the floral meristem, influencing the activity of the floral meristem.

GA regulates multiple pathways affecting flower induction and autonomous pathways, photoperiodism, and stress responses[12]. Gibberellic acid's major activity in the flowering pathway is signaling 'to induce fertility'. Gibberellins also affect the rate of cell division and dormancy breaking in seeds and buds, and induce growth at lower temperatures[11]. Ethylene plays a large role in the regulation of flower initia-

tion; it delays flowering in Arabidopsis[13]. ABA is considered a 'floral repressor' due to its role in drought stress mechanisms and the ability to delay flowering. The hormone SA signals proteins to induce floral buds in response to abiotic stress. The hormone JA affects floral maturation[8].

Transcriptomic analysis

Floral bud and flower differentiation and development are a combination of various physiological and biochemical processes. There are multiple hypotheses of how flowering is affected by hormones; the hormonal balance hypothesis[14,15], the hormone regulated flower gene expression hypothesis[16], and the hormone-signal-regulating flower bud differentiation hypothesis[17]. Combining endogenous and exogenous hormone studies with transcriptomic data analyses could potentially produce a better understanding of the role of hormones in flowering. Transcriptomic analysis is a dynamic representation of the cellular state. Generally, transcriptome studies identify the differentially expressed genes under different conditions, leading to a new understanding of the genes or pathways linked to the specific organ or conditions. In this section, we focus on the relationship of plant hormones to flowering as revealed by transcriptomic studies in fruit and nut trees.

Hormonal signaling's role in flowering is regulating the *FLOWERING LOCUS C* (*FLC*), *CONSTANS* (*CO*), and *FLOWERING LOCUS T* (*FT*) genes. For example, SA is involved in regulation of the transcription of *CO*, *FLC*, *FT*, and *SOC1* (*SUPPRESSOR OF OVEREXPERSSION of CO 1*) in *Arabidopsis*[18]. In *Arabidopsis* under light stress SA promotes the transition from the vegetative to the reproductive phase. In non-stressed Arabidopsis plants, it regulates flowering timing; it acts as a negative regulator of the floral repressor genes *FLC* and other components of the autonomous flowering pathway[18]. A similar effect of SA on flower induction was observed in the short day plant *Pharbitis nil* under stress[19]. The expression of *PnFT2* (orthologous of flowering gene *FT*) during stress conditions suggests its role in stress induced flowering[19]. The CO protein responds to increased day length and activates the expression of the *FT* gene. The *FT* gene produced FT protein is transported to the shoot meristem via the phloem where it activates the expression of the genes involved in flower induction (*AP1* and *LFY*)[20]. The *FT* also activates *SOC1* which initiates *LFY* (*LEAFY*) transcription and translation. The floral development gene *LFY* is thought to be a master regulator[21]. The *LFY* and *SCO1* genes are activated by GA[20,21]. In pecan, bearing shoots experienced a sharp increase in *LFY* gene expression during the 'off-year' in July, whereas nonbearing shoots showed almost no change in *LFY* gene expression during the whole fruiting season[20]. Recently, pecan homologs *leafy* (*CpLFY*), *apetala1* (*CpAP1*), and *flowering locus t* (*CpFT*) were studied to observe if their expression changed or varied by PGRs application over a 2-year study[22]. They observed significantly lower expression of *CpLFY* in shoots treated with GA, whereas higher expression in AVG treated shoots. The differences in expression of *CpLFY* and *CpAP1* based on PGR applications supported the idea of the role of plant hormones and these gene expressions in pecan pistillate flower initiation[22]. Further research is needed to understand the effect of PGR and ratios of *CpLFY* and *CpAP1* in buds of trees with high Alternate bearing and low Alternate bearing[22].

Auxin is another plant hormone that controls the initiation of flower primordia. For example, an increase in H3K9ac (Anti-histone 3 lysine 9 acetylation) at *LFY* and *FIL* (*FILAMENTOUS FLOWER*) loci by auxin application, leads to an increment in mRNA accumulation of both genes and promotes floral primordium initiation in Arabidopsis[23]. When not treated with auxins, the transcription of *LFY* and *FIL* in inflorescence apices is inhibited by the transcriptional co-repressor TOPLESS (TPL) and the histone deacetylase (HDA19), which binds to the MP (MONOPTEROS)-bound site of these genes. The MP proteins are required for the formation of flowers by modifying the chromatin structure[23]. In *Arabidopsis*, abnormal inflorescences and flowers were observed in pin-formed mutant pinl-1 and mutant pin1-2 (pin1 gene's primary function is transport of auxin in inflorescence axis). The pin-formed mutant pinl-1 which causes the disruption of normal polar auxin transport, and were unable to form floral primordia[9]. However, exogenous application of the auxin IAA (Indole-3-acetic acid) can reverse this and induce floral formation[24]. In sweet cherry, Aux/IAA proteins degrade as the auxin level rises in floral buds, and ARF-like protein(s) may induce the expression of their target LFY gene. The ARF transcription factors appear to have a significant role in defining and sustaining unique auxin responses in certain tissues or at different stages of development. Many ARF-like genes (ARF1, 2, 3, 4, 6, 8, and 18) have recently been revealed to show differential expression during floral bud development, implying that these genes are involved in sweet cherry flowering induction and organogenesis[25].

Most of the transcription factors in the complex gene regulatory network that control plant flower development belongs to the MAD S (MINICHROMOSOME MAINTENANCE1, AGAMOUS, DEFICIENS and SERUM RESPONSE FACTOR)-box family[26]. The MADS-box family genes are divided into two groups according to type of gene domain; Type I (M-type: include α, β, and γ subfamilies) and Type II (MIKC type: include MIKC* and MICKC subfamilies). In pecan, the phylogenetic tree revealed a high expression of type-II genes at full bloom stages, while six type-I genes were not expressed in this stage[27]. Similarly, higher expression of MADS-box transcription factors (1 and 12 genes) in the female and male inflorescence suggested that role of MADS transcription factors in the development of pecan flowers[28]. The pecan floral, MADS-box, hormones (auxin and CTK) related genes, and *AP2* (*APETALA 2*) genes were demonstrated to have high connectivity in these pathways[27]. Furthermore, 16 MADS-box genes were hubs because of their high connectivity in the network. The hub genes with the highest correlation in candidate modules were related to photoperiod (*COL1*, *COL2*, *PHYA*), GA (*DELLA*- 2 genes), and flowering (*HD3A*, *LHY*) indicating that the photoperiod and GA pathways are important factors regulating pecan flower development.

The genes related to hormone signaling produced significantly different expressions during the different bud and flower stages in pecan[27]. Similarly, significant differences in gene expression related to CTK, IAA, and GA pathways have been identified at the different bud differentiation and flower

stages between two apple varieties[12]. The up-regulation of genes related to CTK, ABA, SA, and JA biosynthesis in buds of 'Qinguan' a profusely flowering variety, indicates their role in floral induction and higher flowering of this cultivar compared to 'Nagafu No.2'[12]. In sweet cherry the enrichment in genes with roles in the signal transduction of auxins, CTK, and ABA suggest that these hormones and their related genes play a role in floral bud differentiation[25].

In pecan, a recent comparative transcriptome analysis revealed a total of 5286 DEGs between catkins and pistillate flowers[28]. A high number of these genes were involved in phytohormone control, particularly in the biosynthesis (Gibberellic acid-2-oxidase: GA2ox and Gibberellic acid-20-oxidase: GA20ox), regulation (*GASA*, *GRF*, *GRAS*), and signal reception (*GIBBERELLIN INSENSITIVE DWARF1*: *GID1*) of gibberellins[28]. The gibberellin dioxygenases enzymes are related to GA synthesis and consist of two biosynthetic enzymes GA20ox and GA3ox, and an inactivating GA2ox enzyme, the most important sites of regulation in the GA pathway[29]. The GAs regulate the development and fertility of flowers by suppressing the function of the DELLA proteins[30]. Generally, DELLA proteins inhibit plant growth[31] and GA receptors such as GID1 enhance the degradation of the transcriptional regulators of the DELLA proteins[32]. The GA contributes to the destruction of DELLA proteins, while lower levels of GA lead to an accumulation of DELLA proteins[31,33,34]. Flowering defects results from a loss of function of any component of GA biosynthesis and signaling[35–37]. For example, the GA1 gene encodes an ent-kaurene synthetase enzyme in the first step of GA biosynthesis. Gibberellin-insensitive1-3 (GA 1-3) mutants which are deficient in GA1 gene either never flower or delay flowering during short-day conditions[36,37].

GA2ox and of GA20ox related genes and *GASA5*, *GASA11*, and *GASA6* gene expression differences revealed that these are involved in pecan flower sex differentiation[28]. Other plant hormones such as CTK and ABA also play a role in flower sex differentiation. The results from a study on *Castanea henryi* suggest that GA, CTK, and ABA have important roles during sex differentiation, whereas the involvement of IAA does not appear to be important[38]. Their results also indicated that GA and ABA are more involved in male flower development (stamen and anther development), while CTK is more active in female flower development (pistil primordium induction). The hormone CTK is considered a 'female hormone' because it exerts significant control of female flower development[38]. Previously, complex patterns of gene expression were discovered throughout strawberry flower development and early stage fruit development, with notable tissue- and stage-specific modulation of gene expression linked to hormone metabolism, transport, and signal transduction[39].

Exogenous Application

Plant hormones exist in two forms, those produced by the plant, known as phytohormones, endogenous, or natural plant hormones, and synthetic hormones known as bio-regulators, plant growth regulators or PGRs[11]. The synthetic PGRs mimic the function of natural plant hormones, regulating plant growth and development. For example, indole-3-acetic acid, IAA, the most abundant form of auxin, indole-3-butyric acid IBA, and 4-chloroindole-3-acetic acid, 4-CL-IAA are natural auxins, while naphthalene-1-acetic acid, 1-NAA, and 2,4-dichlorophenoxyacetic acid, 2,4-D, are synthetic auxins. Similarly, Zeatin, is the most common naturally occurring CTK, while Kinetin is a synthetic analog. However, they have similar structures. Other CTKs, dihydrozeatin, DZ and isopentenyl adenine, iP, are common in higher plants, while Benzyladenine (BA) is a synthetic CTK. The external application of growth regulators can enhance or inhibit the actions of specific plant hormones. The endogenous concentrations have been studied in multiple cultivated tree crops; walnut, olive and pear[14,40,41]. The following section discusses the effects of exogenous applications of plant growth hormones on growth and development of tree crops.

Bud break and flowering

Flowering consists of three stages: induction, initiation and differentiation. During induction and initiation stages, the high GA_3 content has an inhibitory effect, while the high level of GA_4, ABA, and certain CTK may have a positive effect on flower formation in olives[41]. For instance, in pecans GA_s inhibited flowering as its treatment reduced the number of flowering shoots and female flowers per cluster[42]. Similar inhibitory effects of GAs and simulative effect of CTKs on floral induction of perennial trees have been reported by Bangerth[43].However,P-Ca(calcium3-oxido-5-oxo-4-propionyl-cyclohex-3-enecarboxylate; an inhibitor of GA synthesis) and ethephon (an ethylene generator) were more effective in promoting the percentage of terminal pecan flowering shoots and the average number of female flowers per cluster when applied individually rather than in combination[42]. In cashew, treatment of GA_3 led to peak flowering 4 weeks earlier during cool temperatures and therefore might be beneficial in promoting flowering as it led to flower initiation and development[44,45]. However, a concentration dependent inhibition of initiation and delay of flowering was observed in mango treated with GA_3[46]. Similarly, the endogenous GAs, GA_1, GA_4, and GA_7, have been reported to inhibit floral initiation in 'Golden delicious' apple[47].

Some studies suggest that exogenous hormone applications promote flowering in tree crops. For example, in apple, exogenous CTK treatment on spurs during flower initiation increased the flower number and return bloom[46]. Like the CTKs, JA also stimulates floral induction and initiation in apples[48,49]. The bioregulators TIBA (an auxin transport inhibitor), BA (a CTK) treatments had a highly positive effect on pecan flowering when applied in combination with each other and no impact when applied alone[42]. Sprays of 6-BA, 6-benzyl amino purine, a CTK that promotes flower bud formation, applied before flower induction on apple trees indirectly affects the endogenous Zeatin/IAA ratio. It reduced the Zeatin/IAA ratio; alteration of this ratio leads to lower expression of the flowering gene *MdTFL1* (Homologues of *TFL1- TERMINAL FLOWER 1*), resulting in a higher expression of the *AFL1* (*APPLE FLORICAULA/LFY*) at flower initiation and alters the shoot component and growth characteristics, ultimately promoting flower development[50]. In contrast, ABA inhibited apple bud growth when injected into the xylem[48].

This inhibition could be overcome with follow-up injections of BA. These results confirm earlier reports that exogenous applications of CTKs can increase bud-break in apple trees[51].

Dormex®, hydrogen cyanamide (HC), a dormancy breaking agent, produces a more uniform bud break. In pecan, a hydrogen cyanamide treatment before vegetative bud break led to significantly advanced bud break, male and female flower maturity, and nut maturity without any negative effect on nut yield or phytotoxicity[52]. A Dormex® plus potassium nitrate spray applied 4 weeks before bud break on pecan produced earlier, better and more synchronous flowering of the female and male cultivars[1]. A significant higher and earlier bud opening during spring was observed after HC treatments in December in 'Curtis' pecan trees compared to controls during 2014 and 2015[53]. Recently, similar effect of Dormex® has been observed in 'Wichita' and 'Navaho' pecan trees on bud break timing[54]. They observed significant higher bud break percentage in Dormex® treated compared to controls. Even though they did not observed distinction between vegetative and reproductive bud improvement, but observed significant higher yield on 'Wichita' treated with Dormex® during first year of study[54].

Delaying bloom

Bud break and bloom are the stages most sensitive to environmental conditions; particularly spring frosts. Ethephon, 2-chloroethylphosphonic acid, ethrel® has been used to delay bloom and avoid frost or freeze during spring and increase bud survival rate in sweet cherry, plum, apricot, peach, prune, almond, etc.[55–60] (Table 1). Ethephon, a PGR, precipitates ethylene production in the plant tissues reducing cell elongation and crop height[61,62]. Cultivars and tree crops differ in their sensitivity to ethephon. It can delay bloom (almond, peach, apricot, plum, sour cherry, and prune trees) or have no effect on bloom (almond and peach) or have detrimental impact on flower density, fruit set and yield (some peach cultivars) (Table 1). Rates higher than recommended may result in tree injury, such as excessive defoliation, reduced catkin formation and twig dieback. Proper application and timing are essential for a successful response to any PGR[63].

Lateral/axillary buds

There are two types of buds on fruit trees: terminal and lateral buds. A terminal or apical bud, is located at the tip of a shoot, while a lateral bud develops at the base of a leaf blade along the shoot. Pear and apple flower and fruit primarily on terminal buds. The flower/fruit buds in pears can be terminal on long shoots (more than 4 inches) or more commonly on spurs, which are small branches[64]. Syllepsis, branches without a dormant period growing from lateral buds during the same growing season in which the buds are formed could occur when resources, nutrients and water are abundant. The concentrations of auxin and CTK influence sylleptic bud break and branch growth. Auxins produced and translocated from the apical meristem can inhibit subtending laterals and contribute to apical dominance in numerous species, including apple. High CTK concentrations in shoots can reduce the influence of apical meristems inhibiting the growth of axillary/lateral buds. Shoot tips of upright-narrow canopy apple trees had a higher auxin: CTK ratio, and numerically greater auxin concentrations, compared to the spreading-round growth habit of apple[51]. Auxin-CTK interactions may affect a number of processes that regulate bud growth, including apical dominance[51]. Another plant hormone such as GAs, ABA, ethylene is also associated with induction or inhibition of axillary bud growth[65].

The most pecan axillary buds have capacity to produce flowers if allowed to complete development[66]. In pecans, axillary bud growth and its relation to PGRs has been studied by wood[65]. The axillary bud growth induction was observed significantly only in BA and Promalin treatments, whereas no effect of GAs, ABA, IAA application was observed on axillary bud growth and shoot retention. Further, they also studied the different hormones present in axillary buds and shoots[65].

Alternate bearing (AB) and return bloom

Alternate baring, alternating heavy crops, ('on' year) and light crops ('off' year), is common in fruit crops; pecan, apple, citrus, and pistachios. During the 'on' and 'off' year of olive, the level of endogenous GA_3, ABA, IAA, and kinetin-like substances (i.e. CTK) varied significantly in olive[40]. The ABA, IAA and GA_4 levels in olive leaves, nodes and fruits during the induction, initiation and differentiation periods in the 'on' year were lower than those in the 'off' year[41]. Whereas, GA_3 and kinetin content found higher in leaf, node, and shoot tip samples from 'on' year as compared to 'off' year in olive, mango, lychee samples[40,41,67,68]. Further, Baktir et al.[40] reported higher concentrations of GA_3 promoted vegetative

Table 1. Summary of exogenous hormone (Ethephon) studies on growth and development of tree crops.

S. No.	PGRs	Fruit/nut crop	Effects	Reference
1	Ethephon	Sweet cherry	Delay bloom 3-5 days, Reduced spring freeze injury and increased yield	Proebsting and Mills[56]
2	Ethephon GA	Victoria plum	Ethephon and GA delays bloom for 1–13 days, yield double in 1981 and 14 times in 1982 Individual application of GA and ethephon have variable results No detrimental effect on fruit quality or maturity by both regulators	Webster[57]
3	Ethephon	Apricot and Peach	Delayed bloom in both crops Reduced yield in untreated Apricot after frost of −4 °C Less damage in treated peach as compared to untreated Fruit set not affected or slightly increased	Buhan & Turi[58]
4	Ethephon	'Empress' peach	Delayed bloom No effect on Bloom density, harvest date	Ebel et al.[59]
5	Ethephon	Peach and Prune	Delayed bloom Reduced yield in peach In prune, higher yield after treatment year even after frost at full bloom stage, while reduced yield after treatment year when no frost occurred	Crisosto et al.[60]
6	Ethephon	Almond	Not recommended for almond in Mexican warm climate	Grijalva-Contreras et al.[61]

bud growth, while a lower content of GA_3 promoted flower bud formation. The ratio of GA_3 and ABA is associated with blooming and fruit set. A lower GA_3 and higher ABA content favored the flower bud formation in olive[40].

There is a complex relationship between inhibitor hormone content and flowering. The heavy crop load resulted in an increase of inhibitor phytohormones and suppressed the return bloom in the subsequent year. In axillary buds of apple, IAA and GAs showed a strong negative correlation; as the level of IAA increased, GAs levels decreased. A significant reduction in return bloom in fruiting shoots as compared with non-fruiting shoots suggested that the content of GAs was more important for floral inhibition than content of IAA[69]. In an 'off' year, P-Ca (a GA synthesis inhibitor) was highly effective in promoting pecan pistillate flowers when applied in higher concentration (500 mg/L) at the pre-kernel filling stage of nut formation. While it had no impact on return bloom when applied at a post-kernel filling stage, even at higher concentrations[42]. Moreover, strong positive correlations between inhibitor hormones and sugars, between IAA and GA_1, and between GA_3 and GA_7 resulted in the highest rate of return bloom following year in non-fruiting trees[69].

In pecan, the impacts of PGRs on the subsequent season's flowering are complicated. The return bloom of non-fruiting and fruiting shoots of 'Western' pecan varied significantly in response to PGR treatments. Immature 'Pawnee' shoots had no statistically significant differences in response to the same PGR applications. In current season non-fruiting shoots on immature 'Western' trees, a GA_3 application reduced the number of flowers per new shoot in the following season by 88.2%[70]. Similar results were observed in 'Pawnee' by GA_3 and GA_{4+7} application[42]. Similarly, AVG (Aminoethoxyvinylglycine- inhibit ethylene synthesis) increased fruit retention in 'off' years and has a carryover effect in which the subsequent season's 'on' crop load was reduced[42,70–72]. However, ethephon treatment to immature pecan increased both the following season's percentage of new shoots with flowers and the number of nuts per cluster compared with the control trees[42]. On the other hand, in 'Wichita' pecan, ethephon application resulted in better quality crop by reducing nuts number in the excessive nut crop load year[63]. Water investigations with GA_3, ethephon, AVG both increased and decreased pecan production through effects on return bloom. The effect of GA_3, P-Ca, TDZ (thidiazuron) application has been studied on 12-year-old 'Western Schley' pecans during a two-year experiment[73]. They observed significant variations in the AB index compared to controls[73]. Therefore, these PGRs may have potential for mitigating AB in pecans[73], as is in some fruit crops[70]. These PGRs such as GA_3, ethephon, AVG, can be an alternative for controlling excessive nut production by mechanical methods as mechanical methods may damage trees, making them more susceptible to insects and pests, or damage the nuts[62].

Shoot growth

In the late 1970s, cell elongation inhibitors i.e., paclobutrazol (PBZ, as GA inhibitor), uniconazole (UCZ), and flurprimidol, were the first major discovery of commercially feasible tree growth retardants (TGRs) usable on a large scale. PBZ is more potent and required in relatively low concentration for inhibition of shoot growth as compared to the other TGRs. The primary effect of PBZ on trees is reduced tree height by reducing internode elongation, resulting in greener and more compact growth. The rise in chlorophyll concentration per unit leaf area due to compact growth by PBZ makes the leaves greener[74]. The increased chlorophyll per unit leaf area could also increase photosynthetic activity. The PBZ application significantly reduced the growth of fruit trees such as cashew, peach, apricot, pecan, and mango[75–79] (Table 2).

The major principle in high density plantings is controlling vegetative growth for sustained productivity. In pecan, the vegetative shoot growth, yield, and nut size can be controlled by PGRs. Some experiments reported reduced terminal shoot growth, leaf area, and lower yield or nut size after PGR applications[80]. However, Gaash and David[81] were successful in controlling pecan tree shoot growth with PBZ or 'Cultar®'. The trunk of the vigorous 'Mohawk' cultivar was treated in 'off season' ('low' crop year in July and October) and produced rosettes of dark green leaves and less shoot growth with heavy yields in the following season. When trunks of the 'Delmas' cultivar were treated during spring season, growth was vigorous that season but reduced the following two years. Yield and nut size both increased in treated trees during all three experimental years. The PBZ treatments to 'Delmas' trees caused slowed shoot growth and produced higher yields[81]. In another study by Anderson[82] on 12-year-old 'Cape Fear' and 'Desirable' trees, 'Cultar®' did not affect

Table 2. Summary of exogenous hormone (PBZ) studies on growth and development of tree crops.

S. No.	PGRs	Fruit/nut crop	Effects	Reference
1	PBZ	Cashew	Treatment inhibited the taproot growth and promoted the growth of the lateral root, Treated grafts showed normal growth when planted in the field	Misra and Singh[75]
2	PBZ	Cashew	Reduced plant height, canopy spread, and intermodal length An increase in the number of flushes with a yield increase up to 51.78%	Meena et al.[76]
3	PBZ	Mango	Significant difference on the time to flowering, percentage of flowering shoots, and panicle length PBZ at 2,000 mg/L were caused stunting of flushes and panicle malformation	Wongsrisakulkaew et al.[77]
4	PBZ	Peach	Significantly reduced the competing spring shoot growth and resulted in earlier maturity of a greater crop of larger, better quality fruits.	Allan et al.[78]
5	PBZ	Apricot	Significantly reduced vegetative growth Lower total pruning dry weight, shoot growth and trunk cross sectional area of treated trees than controls. Fruit load, crop density and total soluble solids of fruits were not affected by PBZ compared with the control.	Arzani and Rousta[79]

vegetative growth during the first year of the experiment (1986), but reduced the shoot growth and trunk diameter in the second year (1987). Recently, retarded terminal shoot growth and a significant higher number of pistillate flowers on short shoots has been observed after PBZ application on 6-year-old 'Mahan' pecan trees[83]. Similarly, PBZ application on 75-year-old 'Stuart' pecans trees reduced the terminal shoot growth and leaf area during four years after application[80]. However, higher doses can reduce the nut production in pecans[80]. UCZ is a triazole compound related to PBZ[84]. In 'Wichita' pecans (ten-year-old tress), UCZ application resulted in increased in trunk diameter but reduced shoot growth, lateral shoots per terminal, internode length, and leaflets per compound leaf[84]. The reduced shoot growth could be interpreted as the tree's strategy to better support the flower and fruit production, given the ability of short shoots to export more resources earlier to terminal growing flowers and nuts[81].

Perfect flowers and improved sex ratio

Fruit trees can have either perfect flowers, a flower having both male and female reproductive organs, or imperfect flowers, a flower with only one reproductive organ either male or female, pecan, walnut, pistachio have imperfect flowers, while apple, peach, cherry, cashew have perfect flowers. Foliar applications of exogenous hormones increased the number of perfect flowers in cashew increasing yield. This increase in perfect flowers was also observed with foliar application of ethrel, 1-NAA, Kinetin, and GA_3[44,85–87]. The number of male flowers was reduced with NAA applications and increased by GA_3 applications has also been observed in cashew[86,88]. An RNA study showed that a higher active GA content expressed in Chinese chinquapin male flowers compared to female flowers, suggesting the high levels of GA facilitate differentiation in male flowers[38]. Collectively, this suggests hormone application might be useful for inducing male or female flower production in nut and fruit trees. For example, pecan produces more male than female flowers. Induce more female flowers could, theoretically, increase yield.

Fruit set and fruit drop

Synthetic auxin applications, NAA, 2,4-D may prevent fruit drop. Auxin deficiencies in growing fruits leads to abscission. Fruit drop of cashews was improved with[89]. The hormone-specific and optimum concentration-response patterns were evaluated in two cashew cultivars at five different concentrations. Of the five hormones applied, GA_3, IAA, IBA, NAA, and 2,4-D, the GA_3 was most effective at 50 mg L^{-1} as it enhanced the flower production, fruit set, fruit retention, and nut size compared to untreated/ control twigs[44].

In mango, the parthenocarpic seedless fruits had extremely low CTK concentration compared to fertilized fruits with seeds. This low level of CTK correlated with 100% fruit drop of the parthenocarpic fruits at the marble stage[90]. These results suggest that GAs, CTK, and auxins are all involved in mango fruit development. Reduction or lack of any of these plant hormones may result in lower fruit set[90]. Application of these hormones may improve the mobilization of metabolites and fruit set.

Conclusions

In this study, we reviewed the transcriptomic analysis and role exogenous application of plant hormones in bud break, flowering, and fruiting, focusing on pecan. Different plant hormones (auxin, GAs, CTK, ABA, Ethylene, SA, JA) have varying importance in flowering and are often involved in different stages of flowering development beginning with the signaling of transcription relating to flower induction. These hormones not only play a role in the induction and initiation of flowering but are also involved in flower sex differentiation. Responses to exogenous application of plant hormones varied significantly between hormone type, concentrations, and genotypes. Days to flowering are sensitive to the hormone type, while the production of hermaphrodite flowers, fruit set, and nut development is responsive to a specific hormone concentration. Hormones work in a complex network to control flowering. They can enhance or reduce the activity or expression of each other at particular stages. PGRs function in a similar fashion; a complex balance of inter-relationships among PGRs exists. Application of one PGR may be inhibited or stimulated by the presence of another phytohormone or PGR.

Collectively these studies demonstrate that GAs, auxins, ethylene, and CTK influence floral initiation in pecan. Therefore, at least one key process is largely controllable by the action and/or interaction of one or more molecular species of these plant hormones. The timely application of PGRs to a tree might change the endogenous hormonal content of primordia in such a way as to enable control of flowering by pecan farmers. The efficacy and horticultural potential of plant hormones to control pecan flowering stages have not been fully reported despite considerable circumstantial evidence that endogenous hormones are involved in floral initiation processes. Wood[42] also suggested that in pecan, considerable research is required to determine the appropriate bio-regulator mixture, rate, and application timing. However, there are few studies of hormonal application and endogenous content measurement of phytohormones in pecan. By studying the different aspects of plant hormones, we can apply these results to improve pecan flower quality and nut production.

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Conflict of interest

The authors declare that they have no conflict of interest.

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