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Review

3-Chlorophenoxypropionic Acid as a Plant Growth Regulator in Fruit Crops: A Critical Review of Crop-Specific Responses and Unresolved Mechanisms

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Abstract

This critical review examines 3-chlorophenoxypropionic acid (cloprop), its propionamide and Fruitone CPA formulations in peach/nectarine, pineapple, grape, and pecan. A structured literature update supplements known sources, distinguishing chemical identities and overlapping reports. Inspected direct reports span 1960–2026, driven by pineapple; grape and pecan evidence remains historical. Peach thinning responses vary with developmental stage, cultivar, and dose, while effects of tree physiological status remain hypotheses. Pineapple responses include delayed natural flowering, altered fruit maturation, and crown reduction, with protocol-specific efficacy and phytotoxicity. Hormonal measurements in treated peach and pineapple tissues do not establish a causal mechanism. The pineapple physiological experiment reported in a thesis and subsequent article combined delayed flowering with tissue injury and represents one experimental series. Grape evidence comprises one screening report; four historical pecan reports have important formulation, design, and harvest limitations. These crops delimit the review rather than encompass every investigated crop. Incomplete retrieval and citation tracing limit coverage. The synthesis supports chemically explicit, controlled research on unresolved responses and mechanisms. Historical findings do not establish contemporary efficacy, safety, or authorization. No application or dosing recommendations are provided; contemporary validation and jurisdiction-specific regulatory assessment would be required before practical use.

Keywords: chemical thinning; cloprop; fruit abscission; pineapple flowering regulation; plant hormones; synthetic auxin

1. Introduction

First, 3-Chlorophenoxypropionic acid (3-CPA; CAS 101-10-0), also known as cloprop, is a synthetic plant growth regulator with a meta-chloro substituent and a propionic acid side chain [1]. Historical experiments used the free acid, propionamide, salts, and commercial products with differing or incompletely reported compositions. Several peach reports describe Fruitone CPA as a mixture containing 7.9% propionamide and 0.4% free acid [2–4]; that composition is not assumed for every product bearing the trade name. This review focuses on thinning in *Prunus* persica, flowering, maturation, and crown responses in *Ananas* comosus, and the more limited evidence in *Vitis vinifera* and *Carya* illinoensis. These four systems delimit the present review; they are not asserted to be the only crops investigated.

Interpreting this extended record is complicated by two main factors. First, 3-CPA belongs to a structural family, the chlorophenoxyalkylcarboxylic acids, within which closely

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related compounds share abbreviated nomenclature, partially overlapping biological activities, and interchangeable citation histories. Moreover, 4-Chlorophenoxyacetic acid (4-CPAA), 2,4-dichlorophenoxyacetic acid (2,4-D), and related agents carry substantial literature references in fruit-growth regulation and abscission. The abbreviations CPA and pCPA appear different times in the literature for compounds at both meta and para positions and in both acetic and propionic acid forms. Where compound identity is not verified at the level of individual studies, synthesis can by accident combine evidence for structurally distinct molecules with different biological activities. Second, interpretation requires a source-level distinction between 3-CPA evidence and the broader chlorophenoxy and auxin literature. This review organizes the inspected reports by chemical identity, crop response, and experimental limitations; its documented coverage does not establish the absence of earlier syntheses.

Publication coverage differs among crops: the inspected direct reports span 1969–1990 for peach/nectarine, 1960–2026 for pineapple, 1971 for grape, and 1965–1977 for pecan. These are publication years, not continuous experimental series or evidence of contemporary validation in every crop. Formulation, developmental stage, and the limits of the original designs affect how each response can be interpreted. Most peach thinning experiments concern older cultivars and orchard conditions, so their findings do not establish efficacy under contemporary production systems. Physiological measurements also require careful interpretation: Buchanan et al. [5] measured ethylene evolution in treated peach fruit, whereas Min [6] and Min and Bartholomew [7] examined ethylene-pathway indicators in pineapple. The latter two reports share one CPA experiment and combine delayed flowering with tissue injury. These observations support a comparison of measured responses and unresolved mechanisms, rather than a claim that only pineapple has direct hormonal measurements.

In this review, we focus first on the compound characteristics, and then on the crop system. We focus on the available 3-CPA/Fruitone CPA literature about the compound's biological activity across these four crops, and what remains unresolved. The chlorophenoxypropionic acid class has been applied in horticultural crop management since the mid-20th century [8]. Mechanistic interpretation of 3-CPA's activity has historically been placed within Bangerth's hypothesis of correlatively driven abscission (CDA) [9]. The molecular biology of the abscission zone has advanced substantially since the primary 3-CPA trial era; Tranbarger and Tadeo [10] provide a contemporary conceptual vocabulary for abscission events that did not exist when most of the primary literature was generated, but that review does not address 3-CPA-specific measurements and is used here as interpretive context in Sections 6 and 7.

This review is not intended to provide application recommendations, dosing prescriptions, or guidance for current use, as modern validation under contemporary germplasm, production conditions, and applicable regulatory frameworks will be needed before any historical finding could be considered relevant to today's practice. Section Literature Search and Source Selection describes the literature search and source-selection procedure. The review is organized as follows: Section 2 defines compound identity, formulation character, and regulatory context; Sections 3 and 4 present the primary evidence systems (*Prunus persica* and *Ananas comosus*); Section 5 covers the limited evidence from *Vitis vinifera* and *Carya illinoensis*; Section 6 situates the evidence within its interpretive context and introduces the theoretical framework; Section 7 evaluates physiological hypotheses and unresolved mechanisms; Section 8 addresses evidence limitations, modern relevance, and future research; and Section 9 presents the conclusion.

Literature Search and Source Selection

A structured literature search was conducted on 19 October 2026 using CABI Digital Library, AGRICOLA (NAL SEARCH), and PubMed. Ten searches covered different names of 3-CPA, including chemical and commercial names, with and without crop-specific terms. No restrictions were applied to publication year or language.

Original studies involving 3-CPA or identified related formulations in peach/nectarine, pineapple, grape, and pecan were considered for direct analysis. Studies using other regulators, reviews, regulatory documents, patents, and research on other crops were classified separately as supporting sources. Differences in chemical formulation, study design, publication type, and experimental quality were considered when interpreting the evidence.

The 10 searches returned 153 appearances, corresponding to 120 distinct database identifiers and 99 provisional record groups after bibliographic matching. These records were combined with previously collected literature and targeted recoveries. By 22 October, 2026, 69 original PDF documents had been verified: 50 reports relevant to the direct analysis, 18 supporting sources, and one report outside the review scope. Of the 50 direct reports, 32 were linked to database records or an identified component of an aggregate record, while 18 came from previously known sources or targeted recovery. Forty-nine direct reports are cited in the manuscript.

Information was extracted from available original reports, including compound identity, application methods, experimental design, measured outcomes, statistical evidence, and adverse effects. Overlapping publications were identified to avoid treating the same experiment as independent evidence. Therefore, this work is presented as a critical review supported by a documented literature search, rather than a completed systematic review.

2. Compound Identity, Formulation Complexity, and Regulatory Context

2.1. Chemical Identity and Commercial Formulations

First, 3-Chlorophenoxypropionic acid (3-CPA; CAS 101-10-0) [1] is a synthetic chlorophenoxy plant growth regulator defined by a chlorine substituent at the meta position of the phenyl ring and a propionic acid side chain. The free acid and 2-(3-chlorophenoxy)propionamide are related chemical entities that must be distinguished. CAS 101-10-0 identifies the free acid; it does not identify an amide or a commercial mixture. Historical use of abbreviations such as 3-CP and CPA was checked against each original rather than treated as a universal nomenclature rule. In regulatory contexts, the compound is identified by the common name cloprop, the designation used by the US Environmental Protection Agency [11] and retained in the Pesticide Properties DataBase [12]. The chemical names, trade names, abbreviations, and regulatory designations that appear across primary literature are discussed in Section 2.5.

Fruitone CPA is a historical commercial name rather than a unique chemical identity. Bausher et al. [2], Stembridge and Gambrell [3], and Inoue and Bukovac [4] describe a preparation containing 7.9% propionamide and 0.4% free acid. Dalldorf [13] reports the same stated percentages for Fruitone CPA applied to pineapple, and Cobianchi and Rivalta [14] specify this mixture in their 1970 peach experiments. That composition is attributed to these reports, not to every peach, pineapple, grape, or pecan experiment. The PPDB lists historical trade names [12], but a trade-name listing does not establish composition, concentration basis, or equivalence between products. An Amchem supplier acknowledgement alone is insufficient to identify Fruitone CPA or its acid-to-amide ratio.

2.2. Acid and Propionamide Forms

Shaybany et al. [15] compared free acid at 50, 100, and 200 ppm with propionamide at 100 ppm in previously hand-thinned peach trees at pit hardening. In Loadel, both forms increased fruit diameter, but their effects on preharvest drop differed. In Suncrest, the 100 ppm amide treatment shared a statistical group with the untreated control for diameter, whereas the acid treatments differed from the control. The experiment therefore supports cultivar-, concentration-, and outcome-specific comparisons, not general functional equivalence of acid and amide. It also concerns later fruit development rather than the early fruitlet-thinning window.

2.3. Regulatory History and Jurisdictional Limits

In a 2004 final rule, the US Environmental Protection Agency revoked the pineapple food-residue tolerance for cloprop effective 1 February, 2007, following the absence of active registrations for that use and provision for existing stocks [11]. The residue entry covered the acid arising from application of either acid or propionamide; it did not establish biological equivalence or a single commercial formulation. The consulted PPDB version lists non-approval under GB and European Union headings [12], which is not a determination of worldwide authorization. Its individual IARC Group 2B attribution for cloprop was not corroborated against an agent-specific primary evaluation and is not retained here as an established classification. Administrative registration status, hazard classification, and experimental crop response are separate questions; neither historical efficacy nor missing toxicity fields establishes safety.

The US regulatory history described above does not determine authorization in other jurisdictions. Section 4 includes experimental reports from several countries, including recent pineapple publications, but publication of an experiment does not demonstrate registration of the tested product. A current comparison among countries would require dated, official records for each active substance, formulation, crop, and use. Such a comparison was not established by the sources verified for this review; the PPDB fields are retained as secondary information, not a verified global regulatory survey.

2.4. Translocation Classification and the PGR Context

The PPDB describes cloprop as absorbed by foliage but not significantly translocated [12]. Martin and Nelson [16], however, investigated movement of radiolabelled propionamide in peach. These sources differ in chemical form and experimental context. Movement of a labelled CPA compound to an organ is not a measurement of directional endogenous IAA transport and does not establish the mechanism of abscission. Source-specific transport observations are considered in Section 3.

2.5. Structurally Related Compounds

Studying the roles of several structurally related chlorophenoxy and auxinic compounds in the thinning, pineapple-flowering, and abscission literature may result in mistaken attribution if compound identity is not verified by considering studies individually. Throughout this review, direct evidence is restricted to pertinent experiments with acid, propionamide, an identified salt, or an explicitly named CPA product; incompletely reported product identity is labelled rather than resolved by assumption. The principal compounds that require distinction are described below and summarized in Table 1. Structures of some of these compounds are shown in Figure 1.

Table 1. Chemical identity, nomenclature, and evidence-attribution boundaries for 3-CPA and related regulators. CAS 101-10-0 refers to the free acid and is not assigned to propionamide or commercial mixtures. Identity and concentration basis are checked at source level; contextual compounds are separated from direct evidence.

Compound Name and CAS Number	Chemical Form(s)	Role in This Review	Basis for Inclusion or Exclusion
3-CPA free acid (cloprop); CAS 101-10-0	Free acid; propionamide treated as a distinct related entity	Direct evidence; compound under review	-
Fruitone CPA (historical commercial name)	Mixed amide/acid in specified peach reports; composition verified per source	Direct evidence; principal commercial formulation	Trade/formulation name; not a chemical synonym for 3-CPA
Amchem 3-CP CAS 101-10-0	Identity and concentration basis require source-specific confirmation	Direct evidence where compound identity confirmed	Historical free-acid trade name (same active ingredient)
Bidisin Forte CAS 101-10-0	Composition not established by the trade-name listing	Direct evidence in European sources where confirmed	European market name; less frequent in reviewed sources
4-CPAA (4-chlorophenoxyacetic acid; 4-CPA; pCPA) CAS 122-88-3	Free acid	Excluded; compound-identity context only	Para substitution (not meta); acetic acid side chain (not propionic). Appears in Einset et al. [17] and tomato literature [18,19]
2,4-D (2,4-dichlorophenoxyacetic acid) CAS 94-75-7	Free acid; salts	Excluded; contextual only	Dichlorophenoxy; acetic acid moiety; distinct compound class
NAA (1-naphthaleneacetic acid) CAS 86-87-3	Free acid; salts	Excluded; background comparator only	Naphthalene ring system; entirely different structural class
Ethephon (2-chloroethylphosphonic acid) CAS 16672-87-0	Phosphonic acid (ethylene-releasing)	Not 3-CPA evidence; contextual or within-trial comparator (6.5)	No structural relationship to 3-CPA; ethylene-releasing mechanism distinct
CPPU (forchlorfenuron) CAS 68157-60-8	Phenylurea	Excluded; background comparator only	Cytokinin-type compound; different compound class
Picloram	Pyridinecarboxylic acid regulator	Excluded; contextual only	Picloram appears in the excised Citrus pistil bioassay [17]; not 3-CPA evidence
2-(4-chlorophenoxy)propionic acid CAS 3307-39-9	Free acid	Excluded; structural reference only (Table 1)	Para isomer of 3-CPA; not found in reviewed literature; listed to prevent structural confusion

Moreover, 4-Chlorophenoxyacetic acid (4-CPAA; CAS 122-88-3) carries the chlorine substituent at the *para* rather than *meta* position and has an acetic acid rather than propionic acid side chain. Einset and colleagues [17] characterized the effects of 4-CPAA on abscission, alongside picloram and 2,4-D, in excised *Citrus* pistil explants. The abbrevi-

ated forms 4-CPA and pCPA, as they appear in the tomato studies reviewed here [18,19], designate 4-chlorophenoxyacetic acid, not a propionic-acid positional analog. Both abbreviations are treated as referring to 4-chlorophenoxyacetic acid unless a primary source explicitly defines them otherwise. A propionic-acid positional analog does exist, 2-(4-chlorophenoxy)propionic acid (CAS 3307-39-9), but it does not appear in the literature reviewed here. However, it is listed in Table 1 to prevent structural confusion. In all cases, para-position compounds differ from 3-CPA in substitution pattern and, depending on the side chain, in acid class; none is treated as direct 3-CPA evidence.

Also, 2,4-Dichlorophenoxyacetic acid (2,4-D) carries chlorine substituents at both the 2- and 4-positions of the phenyl ring and has an acetic rather than propionic acid side chain. Despite its extensive history in fruit-drop management and in the citrus auxin literature, 2,4-D is structurally and functionally distinct from 3-CPA and is not treated as equivalent to it in this review.

Picloram and 2,4-D were among the compounds tested alongside 4-CPAA in the excised Citrus pistil bioassay of Einset et al. [17]. That study examined inhibition of style abscission, and its radiotracer comparison concerned 2,4-D and IAA. It did not test 3-CPA. Other regulators, such as NAA, ethephon, and CPPU, are retained only where a specific source supports a contextual or comparative role. Domoto and Hewitt [20] studied ethephon in plum; their hormone measurements do not constitute evidence for 3-CPA in that crop.

Abbreviations such as CPA and historical trade names are insufficient to identify a preparation on their own. Bausher et al. [2] report a mixed Fruitone formulation, whereas Burke [21] identifies an Amchem-supplied material as CPA without specifying its acid-to-amide composition. These cases are retained with their different levels of identity information; the unresolved formulation details are not removed by assigning a common manufacturer or brand.

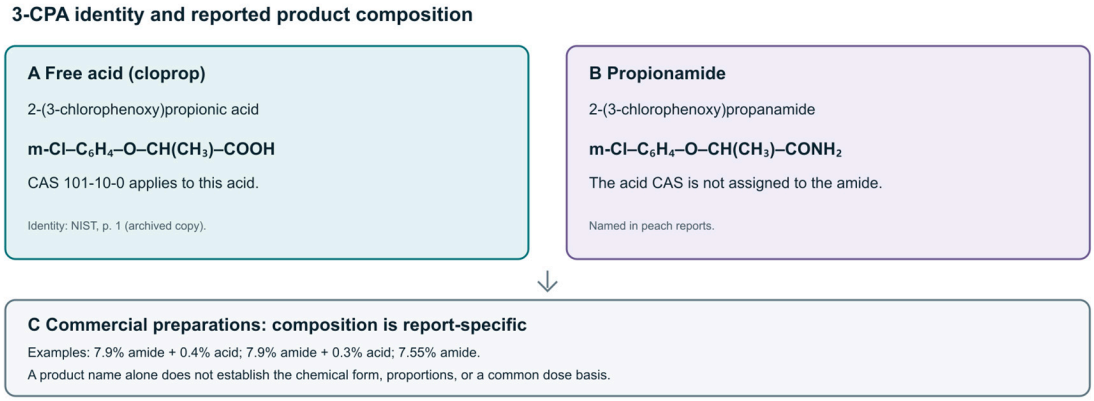


Figure 1. Chemical identity and composition boundaries for the 3-CPA literature. (**A**) Free 2-(3-chlorophenoxy)propionic acid (cloprop; CAS 101-10-0) [1]. (**B**) The corresponding propionamide, which must not be assigned the acid CAS [3,4,16,22,23]. (**C**) Examples of reported commercial preparations: 7.9% amide plus 0.4% acid [2–4], 7.9% amide plus 0.3% acid [24], and 7.55% amide with no acid fraction specified [22]. These reports do not establish a single composition for every product called Fruitone CPA. Percentages are product composition, not treatment concentrations.

2.6. Safety, Toxicology, Residues, and Environmental-Fate Evidence

The historical US residue rule is a regulatory action, not a toxicological experiment. In 2004, EPA retained a 0.3 ppm pineapple tolerance for acid residues arising from application of either the acid or propionamide until its revocation on 1 February, 2007 [11]. The 2001 notice describes a temporary emergency exemption for sodium cloprop on Oahu [25]; EPA subsequently reported an amended Hawaii authorization ending on 2 August, 2002 [11]

(p. 43919). These dated actions cannot establish current registration or the safety of acid, amide, salt, or Fruitone formulations.

Analytical capability likewise differs from evidence of exposure. Pu et al. [26] included cloprop acid among 24 plant growth regulators in a grape LC-MS/MS method (their Table 1), with a 2 ng/mL extract limit of quantitation for cloprop (their Table 3). Cloprop was below that method limit in 50 grape samples collected in Taiwan in 2014 (their Table 7). Treatment histories were not specified for those samples, and the assay did not establish residue behavior after a defined 3-CPA treatment, nor measure propionamide.

The consulted IARC evaluation assigned Group 2B to chlorophenoxy herbicides as a class [27] (pp. 156–160); those pages do not establish an individual classification for cloprop, its propionamide, or a Fruitone product. The PPDB agent-specific attribution [12] is therefore not adopted as established. This attribution limit does not establish safety. Phytotoxicity in the crop trials describes plant injury under particular exposures; it does not quantify human or ecological hazard. The sources verified here do not provide a form-specific assessment of chronic toxicity or exposure from a defined crop treatment. Consequently, no acceptable intake, safe residue concentration, or comparative safety ranking is derived from them.

Environmental fate also remains unresolved by these sources. A residue-detection method does not measure degradation, soil mobility, persistence in water, or metabolite formation. Primary fate and metabolism studies would need to identify the test substance and products, matrix, exposure conditions, and mass balance before acid, salt, propionamide, or mixed-formulation results could be related. These are gaps in the verified evidence, not a finding that such studies do not exist. Current national authorization and formulation-specific exposure and hazard assessments would be required before any practical inference.

3. Evidence on *Prunus persica* Thinning with 3-CPA

3.1. Historical Evidence Base and Compound Identity

The peach literature includes journal articles, institutional bulletins, proceedings, and extension publications from several research programs. The inspected reports span several countries, cultivars, and orchard conditions, but publication format alone does not establish peer review or experimental quality. Evidence is therefore interpreted using the reported design, experimental unit, controls, replication, statistical support, and damage, while multiple reports of an experiment remain distinguishable from independent experiments. Costa and Vizzotto [28] provide historical context on chemical thinning without adding an independent 3-CPA experiment. Supplementary Table S1 summarizes the peach and nectarine responses.

All the trials reported in the literature and reviewed here were conducted under the orchard conditions, pest management regimes, and germplasm of that period, and the findings should be interpreted with caution when extrapolated to modern cultivars or contemporary production systems. Four analytical variables used to organize the peach evidence, developmental timing, tree physiological status, cultivar identity, and dose, emerged as recurring patterns across the primary sources and are used here as a peach-specific, inferential organizational framework. They are not a mechanism model, not a universal 3-CPA response framework, and are not carried over to the pineapple, grape, or pecan literature reviewed in Sections 4 and 5.

The acid–amide comparison of Shaybany et al. [15] concerns fruit size and maturity after hand thinning at pit hardening. Its cultivar- and outcome-dependent results, including different preharvest-drop responses, do not demonstrate equivalence between chemical forms or establish an early thinning window. Concentrations and formulation identities are therefore retained as reported rather than pooled across forms.

3.2. Developmental Timing

Historical reports place effective peach thinning at different developmental stages by cultivar and program. In South Carolina, the most satisfactory Ranger response occurred near 7.5–8.0 mm ovule length [3]; Italian reports found useful responses in some cultivars near 8 mm [23], while the Cardinal series recorded substantial drop at later fruit lengths, with smaller fruit than after hand thinning [22,29,30]. Antognozzi and Salleo [24] also observed sharply different responses among three cultivars treated at 8–9 mm seed length. These data support a cultivar- and protocol-dependent window, not a universal 7–8 mm threshold or independent replication of every report.

Early applications at ovule lengths near 4–5 mm produced reduced or inconsistent thinning in several reports, but later responses also depended on cultivar and protocol. Redhaven required an ovule length of 10 mm or more for satisfactory thinning in the South Carolina trials [3]. That within-program observation does not define the timing window for other cultivars. Inoue and Bukovac [4] tested Fruitone CPA at fruit lengths of 17.3, 22.3, and 28.4 mm in Redhaven. The 17.3 mm treatment did not differ from the control for the deformation measure, whereas the 22.3 and 28.4 mm treatments did. These observations identify an adverse response under the tested cultivar and application conditions; they do not establish a universal deformation threshold above 17 mm.

In the 1970 Ravenna experiments, Cobianchi and Rivalta [14] applied mixed Fruitone CPA to Halford 1 at mean seed lengths of 9.2, 10.9, and 15.2 mm. At 200 ppm on vase-trained branches, the late treatment and hand thinning shared statistical groups for fruit drop (55.06 versus 56.08%) and mean fruit weight, but yield was lower (41.39 versus 63.93 kg per experimental branch; their Table 7, p. 288). Thus, the authors' favorable assessment of late application does not establish preservation of yield. In a separate whole-tree palmette trial, 300 ppm at 10.9 mm increased mean fruit weight but reduced yield (their Table 8, p. 289).

Martin and Nelson [16] treated Paloro peach near 10 mm ovule length and followed material from radiolabelled 3-CPA. Their organ-specific treatments reduced fruit set, and the tracer moved slowly and locally [16]. The experiment did not identify intact 3-CPA at every labelled site, quantify ovule accumulation at a 7–8 mm treatment stage, or establish polar IAA transport. Martin and Nishijima [31] later observed changes in inhibitor- and promoter-like activity in abscising Paloro fruit but identified the inhibitor only tentatively and described the association as correlative.

3.3. Tree Physiological Status

Field observations suggest that crop load and vigor may influence Fruitone CPA thinning responses. The reports discussed below do not isolate their effects from other differences in tree condition, season, or application context.

Bowen and Powell's institutional bulletin [32] reports variable responses to 0, 150, and 300 ppm in Texas from 1969–1970. The authors associated inadequate or excessive thinning at the same nominal rate with different crop densities and attributed tree-to-tree variability to physiological condition, soil, and root problems. Crop load was not assigned as a controlled treatment, and the 1969 Ranger comparison was unreplicated. Beutel et al. [33] separately noted greater thinning in weak than vigorous trees in their California field report; this was a qualitative observation.

These reports support a hypothesis about tree condition as a source of response variability. They do not estimate a crop-load-by-dose interaction or demonstrate that physiological status has a larger effect than dose. No independent controlled replication of that specific hypothesis was identified in the inspected reports.

3.4. Cultivar Identity

Cultivar identity affects both response magnitude and developmental timing, but poor commercial performance must be distinguished from absence of thinning. Cardinal was difficult to thin in some early programs [3,22]. The Cardinal reports nevertheless describe timing-dependent fruit drop, size penalties relative to hand thinning, and foliar damage [22,29,30]. The 1976 report by Morini et al. [29] republishes the first-date 1971 3-CPA arms and controls of Morini et al. (1971) [22] and the 1972 3-CPA campaign of Morini et al. (1972) [30]. These are two campaigns represented across three reports, not independent replications for each publication. The 1971 and 1976 reports give different significance letters for the same 1971 300-ppm versus hand-thinning contrast; statistical equivalence to hand thinning is therefore not inferred. The 1972 reports also differ in stated branch allocation, tree age, and some letters.

"Ranger" thinned satisfactorily at 150 ppm in South Carolina [3] and at 100 ppm in Italian conditions [23]. These observations describe different regional trial conditions and do not isolate a cultivar-by-concentration interaction. "Redhaven" showed a later effective ovule-length window than "Ranger" within the South Carolina trials [3]. This within-program comparison does not establish a universal threshold across programs. The broadest single cultivar screen in this record was the Italian survey by Fideghelli and Monastra [23] across 11 cultivars at four locations, which identified "Ranger", "Redhaven", and "Vivian" as responsive, and they reported useful responses near 8 mm ovule length in responsive cultivars, while other cultivars showed variable or absent response.

In New Jersey, Bausher et al. [2] evaluated five cultivars, including "Rio Oso Gem": "Jerseyqueen" and "Redhaven" were difficult to thin, while "Sunhigh" and "Sunqueen" thinned more readily. Bausher et al. also reported that shading increased thinning effectiveness in the reported comparisons, an effect tentatively linked to breakdown (citing acid-form work) or fruit development and vigor; no mechanism was established for the mixture [2]. Collectively, cultivar identity is a well-documented determinant of 3-CPA thinning outcome within the historical *Prunus persica* germplasm; the mechanistic basis for differential response was not established in these trials.

Ferreira et al. [34] provide additional Redhaven evidence from 1969 and 1970 in Buenos Aires. Their mixed Fruitone preparation contained 7.9% propionamide and 0.4% acid; 150, 300, and 450 ppm referred to commercial product, not to either ingredient separately. Four single-tree replicates per treatment were applied near a 9 mm seed length. Reported thinning was 31% and 30% in controls and 46% and 45% at 150 ppm in the two seasons. The published significance statements concern size-related comparisons and do not establish superiority of one concentration for every outcome. Mild defoliation and transient terminal bronzing occurred at 450 ppm. These observations require the stated formulation, cultivar, season, and statistical scope.

Additional original reports broaden the peach comparison without producing a common treatment effect. Cobianchi [35] compared mixed Fruitone with ethephon and hand thinning in Fortuna and Halford 1; Gerin and Giulivo [36] reported two Collins seasons but did not specify product composition. Retamales et al. [37] tested propionamide against CGA 15281, hand thinning, and an untreated control in two nectarine cultivars and found variable thinning with foliar injury. Svoboda and Bažant [38] compared Fruitone and Ethrel in two peach seasons, with uncertain replication in 1974. Bazzocchi and Cristoferi [39] compared 3-CPA with CEPA and hand and mechanical thinning in Vivian but included no untreated control. Within-study contrasts are described by cultivar, season, experimental unit, and outcome; effects from different agents or chemical forms are not pooled.

Cobianchi and Rivalta [14] found less fruit drop than after hand thinning in Dixired, Coronet, S. Anna Balducci, and Redhaven in 1970. Southland had similar drop to hand

thinning but substantial foliar injury, including about 30% leaf fall, and reduced flowering scores the next spring. Its lower numerical yield was not statistically separated from hand thinning (their Tables 4–10, pp. 286–291). The assigned units were branches in vase-trained trees and whole trees in the Halford palmette trial; branch replicates do not establish independent trees. This 1970 harvest differs from Cobianchi's 1971 experiment [35], although continuity of orchard or trees is unresolved.

3.5. Dose-Response Behavior

Dose response in the reviewed peach trials was not consistent across experimental contexts, and no context-independent effective concentration can be specified from the available evidence. Stembridge and Gambrell [3] documented adequate thinning of responsive cultivars at 150 ppm in South Carolina; Fideghelli and Monastra [23] reported responses in responsive Italian cultivars at 100 ppm; and Beutel et al. [40] identified 300 ppm as the optimal single-spray concentration in California multi-variety conditions. Cultivar, developmental stage at application, and tree condition varied alongside concentration across these programs. These between-program differences do not isolate the contribution of any one factor to the observed dose response.

Phytotoxicity constrains the response range: overthinning and defoliation occurred after 300 ppm at early stages in the South Carolina trials [3]. Buchanan et al. [5] measured ethylene evolution in field-treated peach fruit. In a separate detached-fruit assay, the rise after CPA treatment showed a lag of more than 48 h, unlike the rapid response to Ethrel (their Figure 4, p. 783). That laboratory time course does not establish the field abscission mechanism or a general application window. Stembridge and Gambrell [3] and Bausher et al. [2] also reported that spray additives altered thinning at a given nominal dose.

A pooled meta-analysis of peach thinning was not supported at this cut-off. Reports use different denominators for fruit drop, untreated or hand-thinned controls, whole trees or branches nested within trees, and acid, amide, or mixed commercial formulations. Variance estimates by assigned unit are often unavailable. The first-date 1971 Cardinal arms occur in both the 1971 [22] and 1976 [29] reports, while the 1972 arms occur in both the 1972 [30] and 1976 [29] reports. Statistical letters and multiple fruit counts do not supply independent treatment-effect variances. The evidence is therefore synthesized by campaign and contrast, including fruit size, yield, and phytotoxicity alongside thinning.

4. *Ananas comosus*: 3-CPA Responses in Flowering Regulation, Maturation Delay, and Crown Development

Ananas comosus (pineapple) provides an important independent body of evidence for 3-CPA/Fruitone CPA/cloprop activity across three biologically distinct developmental aims: inhibition of natural-differentiation flowering, fruit maturation delay, and crown-size reduction. These three developmental processes are physiologically different from the stone fruit abscission process documented in *Prunus persica*. The pineapple evidence reviewed here spans multiple countries, cultivars, and protocols in reports published from 1960 to 2026. The most extensively documented cultivar is "Smooth Cayenne"/"Cayena Lisa"; all dose-response observations are cultivar- and protocol-specific. This section is organized by biological target.

4.1. Inhibition of Natural-Differentiation Flowering

Natural-differentiation flowering (NDF) in pineapple refers to the spontaneous transition of the vegetative shoot apex to floral differentiation, triggered by low nocturnal temperatures or other environmental cues and agronomically undesirable when it occurs outside planned harvest windows. Multiple research programs in Mexico, Australia, and

Brazil have documented that 3-CPA/Fruitone CPA inhibits this floral transition when applied during the vegetative phase [41–45]. These observations span more than 3 decades and at least four independent programs, establishing NDF inhibition as the most extensively documented 3-CPA response target in the reviewed pineapple literature.

Natural-flowering studies used different products, concentration bases, application volumes, frequencies, cultivars, and environments. Their results do not establish a common effective interval or a general phytotoxicity ceiling. In the recent MD2 factorial study, the 75 and 128 mg L^{-1} marginal means differed from control at the April census, with a reported CPA-by-pruning interaction [46]. Rebolledo et al. [42] found no significant difference among application times under their specific conditions.

Pérez-Romero et al. [45] reported 91–97% NDF inhibition across three cultivars, "Smooth Cayenne", "MD2", and "Cabezona", at three planting dates and two planting densities in Tabasco, Mexico. This is the most multi-factorial NDF inhibition study in the reviewed literature and includes MD2 response to 3-CPA for this target. The results must be interpreted within the specific humid-tropics environment of that trial. Scott [43], reporting a multi-year trial series in subtropical Australia (1988–1992), documented inhibition of precocious fruiting in sucker growth, a naturally triggered flowering form, with 50 ppm as the most effective single-treatment dose under Australian conditions; the inhibitory effect was limited in duration to approximately 10 months before a subsequent NDF wave occurred, a temporal limit that constrains interpretation of any single-protocol result as providing year-round flowering control. Cunha et al. [41] documented NDF inhibition in the cultivar "Pérola", extending this evidence beyond "Smooth Cayenne" and "Cayena Lisa" to a second commercially important Brazilian cultivar.

Further reports qualify the range of natural-flowering responses. Hernández-López et al. [47] found separation from control in MD2 at their intermediate treatment but not in Cayena Lisa; their methods and conclusion conflict on the dose unit, so no operational dose is inferred. Rabie et al. [48] reported reduced winter flowering of Queen pineapple with acid-described Swelpine before early May in KwaZulu-Natal, together with injury at the initial high product volume and season-dependent crop effects. These results are specific to each location, cultivar, product, and experimental unit.

Gowing and Leeper [49] tested the free acid in a structure-activity screen in pineapple. The 3-chloro compound produced floral forcing from 6 mg per plant in the reported range; the same table also records inhibition at 60 mg per plant and toxicity. This is an exploratory, dose-dependent response profile, not a selective or safe protocol. It must be distinguished from later studies of delayed natural flowering, with their different doses, formulations, and application conditions.

4.2. Fruit Maturation Delay and Quality

Fruit maturation delay and associated modification of fruit quality parameters constitute a second biologically and agronomically distinct response target, documented in Brazil and Mexico across multiple cultivars. This target involves post-anthesis developmental modulation rather than prevention of the floral-initiation transition, and the dose protocols and experimental designs characteristic of the maturation-delay literature differ from those used in the NDF inhibition studies above.

Fahl and Franco [50] reported increases of 20–36% in fruit weight in "Smooth Cayenne" pineapple treated with Fruitone CPA (sodium salt form) in a randomized complete block design in Brazil; crown weight was concurrently reduced and maturation parameters shifted. Rebolledo-Martínez et al. [51] documented that a single Fruitone CPA application at 900 mL ha^{-1} delayed maturation in the cultivar "Cayena Lisa" harvested in spring in Mexico, with associated fruit quality changes. The dose and protocol of that study apply

specifically to the maturation-delay target under those conditions and are not transferable to NDF inhibition protocols or other production environments. Oliveira et al. [52], working with the cultivar "Pérola" in Brazil over a 2022–2023 trial period, examined pre-harvest 3-CPA application and documented effects on fruit development, crown size, and post-harvest quality; this study extends the maturation-delay and crown-modification evidence to "Pérola" under contemporary Brazilian conditions, a cultivar not represented in the earlier literature regarding maturation delay. Collectively, these papers document a maturation-modulating activity for 3-CPA in pineapple that is cultivar-specific, protocol-dependent, and biologically distinct from both NDF inhibition and crown reduction.

Soler [53,54] reported pineapple fruit-growth and maturation comparisons in Côte d'Ivoire, but later harvest and ethephon timing complicate attribution of exportable-yield differences; overlap between the 1985 and 1990 reports has not been resolved. Othman et al. [55,56] described sodium 3-CPA propionate treatment of Gandul pineapple before cold storage in two closely overlapping reports. Lower internal browning at the higher tested concentrations did not establish uniformly better sensory acceptance. These acid/salt and postharvest observations should not be combined with propionamide or floral-inhibition effects.

4.3. Crown-Size Reduction

Crown-size reduction is a third documented 3-CPA response in pineapple, with evidence across cultivars "Smooth Cayenne", "Pérola", and "Giant Kew" in South Africa, Brazil, and India. Crown size affects post-harvest logistics and commercial handling costs in some pineapple production systems; this review documents the evidence for this response and does not evaluate its practical utility.

Fahl and Franco [50] observed concurrent crown-weight reduction alongside fruit-weight increases in their "Smooth Cayenne" trial. Vieira et al. [57] applied Fruitone CPA (7.5% formulation) at 150–200 ppm to "Smooth Cayenne" in Brazil and documented reductions in crown length and weight. Fruit weight was not significantly affected in that study, but the authors attributed the null result to drought conditions during the experiment. Imchen et al. [58] reported that 100 ppm cloprop (3-CPA) reduced crown size, improved several fruit quality parameters, and extended post-harvest shelf life in the cultivar "Giant Kew" in Nagaland, India. That study used a methanol solvent carrier, a methodological detail relevant to cross-study protocol comparisons. Oliveira et al. [52] extend the crown-reduction evidence to the cultivar "Pérola" under contemporary Brazilian conditions, as noted in Section 4.2. Across these studies, crown reduction was documented under geographically and cultivar-diverse conditions; the biological basis for this response was not characterized in the reviewed studies.

Dalldorf [13] tested Fruitone CPA was specified as a mixture of 7.9% propionamide and 0.4% free acid after flowering in "Smooth Cayenne" in South Africa. At 0.2 mg/L (reported as active ingredient), plant-crop yield was 72.17 versus 60.86 t/ha for the water control; at 0.5 mg/L, yield was 80.89 t/ha, while crown mass fell to 15.41 from 20.03 g and crown leaves showed injury (their Table 1, p. 17; their Figure 2, p. 18). The percentage of first-grade fruit did not differ significantly. Replication and the LSD calculation were not described, and the abstract's claim of a significant fruit-length response at 0.2 mg/L conflicts with their Table 1. The study therefore adds a local yield and crown-quality contrast for a mixed formulation, without establishing a general dose recommendation or an effect on floral initiation.

Fruitone-related damage also belongs in the benefit–harm account. Rabie et al. [59] associated product application with fissures and interfruitlet corking in a multifactorial pineapple investigation. The original did not report product composition or tabulated

variances for the relevant graphs, so these observations are qualitative and cannot establish a precise dose–damage effect across products.

4.4. Physiological Interpretation and the Ethylene Dilemma

Min [6] and Min and Bartholomew [7] measured ethylene production, ACC oxidase activity, and MACC in treated Smooth Cayenne pineapple. The thesis experiment 5.4 and article experiment 4 describe the same CPA experiment initiated in 1994, not independent replication. In stem tissue sampled 31 days after 2.5 mg per plant, ethylene production, ACC oxidase activity, and MACC were elevated; ACC did not differ significantly in the article table. The treatment also caused leaf injury and death of the apical meristem in 6 of 17 plants, while flowering was followed in a separate subset of eight plants. The biochemical measurements therefore require interpretation alongside tissue injury and the sampling design.

The two reports differ in the duration of flowering observation. The thesis recorded no visible inflorescence among eight plants receiving 2.5 mg per plant by the end of April 1995 [6]. The article followed the plants until 30 August: all eight had visible inflorescences, with a mean interval of 282 ± 6 days compared with 137 ± 2 days in controls [7]. The later report thus documents delayed flowering rather than permanent suppression. The coexistence of elevated ethylene-related measurements, delayed flowering, and injury does not identify a causal pathway. A contrast with the flowering-inducing use of ethephon does not isolate the mechanism of CPA.

Field reports and the pineapple physiological experiment address different exposure conditions and developmental endpoints. Localized application in pots, with material tissue injury, does not establish a field rate or a general mechanism. Future experiments should separate compound identity, dose and delivery, tissue injury, ethylene production, downstream signalling, and the timing of floral differentiation. This distinction also provides a basis for comparing the pineapple measurements with ethylene observations in treated peach fruit [5] while keeping organ and developmental contexts separate.

5. Evidence from *Vitis vinifera* and *Carya illinoensis*

5.1. Vitis vinifera

The directly inspected grape report in the present corpus is a systematic chemical screening study by Weaver and Pool [60], conducted at the University of California, Davis, in two *Vitis vinifera* cultivars, “Carignane”, a compact-cluster wine grape, and “Thompson Seedless”, a seedless table grape. That study tested more than a dozen compounds at four phenological stages; the data drawn on in this review are restricted to the 3-CPA treatment rows. The methods identify the supplied material as 3-chlorophenoxy-alpha-propionamide rather than a verified commercial Fruitone formulation.

Weaver and Pool [60] evaluated six clusters per treatment by dipping them in solutions containing 3-CPA propionamide, ethanol, and surfactant and analysed the data by ANOVA and Duncan comparisons. At bloom, Carignane had 179 berries per control cluster: 88 at 10 ppm, 7 at 100 ppm, and 2 at 1000 ppm. Thompson Seedless had 272 in the control, 111 at 10 ppm, and zero at 100 or 1000 ppm. Post-set response differed between cultivars: Carignane treatments did not differ significantly, whereas the Thompson Seedless table shows reductions. High concentrations and several effective treatments were associated with shot berries, rachis or peduncle callus, cracking, or cluster death. These cluster-dip observations are not a validated whole-vine production protocol.

The screening design, cluster-level unit, delivery method, and associated injury limit extrapolation to whole-vine applications and commercial outcomes. The experiments do not identify whether altered berry number resulted from changes in pollination, embryo

development, or abscission; those possibilities should not be presented as measured mechanisms. The grape response is therefore considered separately from peach fruitlet abscission and pineapple developmental responses. Neither similar treatment timing nor reduced fruit number establishes a shared mechanism across crops.

5.2. Carya illinoensis

The pecan corpus includes four directly inspected reports rather than a single thesis: Amling and Dozier [61], Hinrichs [62], Hopfer [63], and Burke [21]. They describe historical experiments at Auburn and in the Oklahoma research program. The reports differ in cultivar, season, application stage, design, and identity information and cannot be counted as four interchangeable independent efficacy trials. They include two theses and two proceedings reports, for which peer review is not established.

Amling and Dozier [61] explicitly identify propionamide in Stuart and report concentration studies in 1964: set was 52%, 26%, 12%, and 2% at 50, 100, 150, and 200 ppm, respectively, compared with 57% on untreated branches. These are reported set percentages, not final yield or percentages of thinning. The one-page progress report does not specify sample size or statistical tests and describes injury at higher concentrations. Hinrichs [62] reports strong thinning and phytotoxicity in a 1969 branch-spray experiment; the controls by application date, unit structure, and statistical reporting are incomplete, and treated Western sectors produced smaller nuts in the descriptive harvest comparisons.

Hopfer [63] studied Western in 1970 using sectors allocated within trees. At the earliest application, 160 ppm produced 231 dropped nuts out of 400 by 36 days compared with 128 out of 400 in controls. The reported analysis supports an overall early treatment effect and a reading-date interaction; it does not establish every pairwise dose comparison. Later applications produced little additional drop. The methods call the material CPA without establishing its commercial formulation. These observations expand the timing evidence but do not establish a general efficacy or safety recommendation.

Burke [21] reported a 1976 field trial at Sparks, Oklahoma, using Wichita and Western. CPA was supplied by Amchem, but the methods do not establish the Fruitone brand or an acid-to-amide composition. Concentrations of 0, 75, 100, and 150 ppm were applied to tree sectors in mid-June, about 5 weeks after pollination, with four replications per treatment and cultivar and 100 marked nuts per plot. Wichita used a completely randomized design and Western a randomized complete block design. Cultivar age, coadjuvant, and other conditions differed, limiting a causal comparison of varietal susceptibility.

In Wichita, cumulative drop at 5 weeks was 15.50% in controls and 28.25%, 41.00%, and 60.50% at 75, 100, and 150 ppm, respectively; the final means were separated by the reported LSD test [21]. In Western, 26.75% in controls and 47.00% at 150 ppm remained in the same statistical group. The thesis describes scab and background drop in Western, and the cultivar contrast is also limited by differences in tree age and treatment conditions. Foliar injury occurred at higher CPA concentrations. Frost and animal damage prevented harvest evaluation, so no nut-yield or quality benefit was established. Similar timing of abscission in peach and pecan does not demonstrate a common causal mechanism.

5.3. Limits of Cross-Crop Transfer

The directly inspected grape screening report [60] and the four historical pecan reports [21,61–63] show that these crop systems have a smaller and differently structured evidence base than peach and pineapple. Pecan is not supported by a single source, but the additional reports do not establish uniform efficacy, formulation equivalence, harvest benefit, or a shared mechanism. Cross-crop comparison therefore concerns the range of

measured developmental responses and the limits of each design, rather than validation of one crop by another.

The response variables themselves make clear what the cross-crop record does and does not show. In grape, the response is berry-set reduction during a bloom-window application. In pecan, it is nut abscission during early nut development. In peach, it is fruitlet abscission within the ovule-length window. In pineapple, it is inhibition of natural-differentiation flowering, delay of maturation, or crown reduction depending on the target. These are distinct biological phenomena evaluated in different organs, at different developmental stages, and under different environmental regimes, and they document distinct developmental outcomes following historically labeled CPA treatments without establishing chemical equivalence or a shared mechanism.

6. Evidence Boundaries, Related Compounds, and Interpretive Context

Sections 3–5 focused exclusively on experiments using 3-CPA or its verified forms: however, thinning and abscission research is much broader, using similar plant systems and other plant growth regulators. This section establishes how these related studies complement the 3-CPA record and defines their boundaries.

6.1. Related Compounds and Their Interpretive Role

The literature regarding chlorophenoxy and phenoxycarboxylic acids contributes to this review in two limited ways. First, it defines compound-identity boundaries by showing that molecules structurally related to 3-CPA also produce similar biological responses. Second, it provides physiological context for the kinds of responses that auxinic compounds can provoke in abscission tissues. Other auxin-class and cytokinin-type compounds, including NAA and CPPU (forchlorfenuron), appear in background roles in several of the reviewed papers.

Einset et al. [17] used excised Citrus pistils as an in vitro bioassay and tested inhibition of style abscission by several auxins, including 4-CPAA, picloram, and 2,4-D. The radiotracer comparison of transport and metabolism concerned IAA and 2,4-D, not every compound in the bioassay. The study did not test 3-CPA or fruit thinning in an intact crop. Likewise, the tomato studies [18,19] concern the chemically distinct para-chlorophenoxyacetic compound class. These sources provide context for compound specificity and experimental scope, rather than direct evidence for 3-CPA efficacy or mechanism.

Domoto and Hewitt [20] treated Japanese plum (*Prunus salicina*) with ethephon and measured growth-promoting activity in chromatographed seed extracts using a wheat-coleoptile bioassay. The observations concern auxin-like activity rather than chemical identification of an auxin or a direct transport measurement. The authors proposed an effect on auxin transport but could not distinguish cause from consequence. Ethephon is chemically distinct from CPA; this study supplies physiological context, not CPA-specific evidence in plum.

6.2. Correlatively Driven Abscission as a Hypothesis

Bangerth [9] proposed correlatively driven abscission (CDA) as a model in which dominant fruits or nearby shoot tips suppress IAA export from subordinate fruits. In the proposed sequence, reduced basipetal auxin transport through the abscission zone permits greater ethylene sensitivity and subsequent separation. The model invokes interactions between auxin transport pathways, including inhibition at their junctions. Much of the supporting discussion concerns apple. Its application to CPA-treated peach or to other crop systems remains a hypothesis requiring compound- and system-specific testing.

Bangerth [9] provides a secondary mechanistic framework for interpreting fruitlet competition and abscission. The doctoral source underlying some of its nectarine observations was not available for direct inspection. Findings attributable only to that unavailable source are therefore not treated here as directly verified experimental evidence. The review is used to frame hypotheses without converting its secondary account into proof of a CPA transport mechanism.

Extractable hormone pools describe tissue concentrations at a sampling time and do not, on their own, establish the rate or direction of cell-to-cell IAA movement. Measurements of a labelled applied regulator are also distinct from assays of endogenous polar IAA transport. No direct transport-flux measurement resolving the CPA mechanism was identified in the original sources inspected for this review. This boundary concerns the evidence examined; an unavailable source is not evidence that no such experiment exists.

Developmental-stage dependence and observations of variable thinning with tree condition provide reasons to test a competition-based explanation in peach. They do not establish CDA as the mechanism of CPA action. Evaluating that proposal requires measurements of auxin transport and the timing of growth arrest and separation under identified CPA treatments, together with interventions that distinguish a cause from a response to incipient abscission. Such a causal test was not established by the original CPA experiments inspected here. Section 7 separates those observations from the proposed explanations.

6.3. Contemporary Abscission-Zone Biology as Interpretive Context

Modern plant biology characterizes fruit abscission at the molecular and cellular level as a precisely regulated sequence at the abscission zone, involving ethylene-dependent signaling, cell-wall loosening, programmed cell separation, and the activity of peptide-receptor systems, including the IDA-HAE/HSL2 module [10]. This contemporary framework describes the molecular events that occur during competent abscission and set the basis to design experiments that could characterize how an applied compound might trigger those events. The framework does not address 3-CPA specifically: The article by Tranbarger and Tadeo [10] is a comprehensive review of abscission-zone biology in general, not a study of 3-CPA action.

What the contemporary framework does not provide, within the literature reviewed here, is direct evidence about how 3-CPA triggers or is involved in any of those molecular events. No reviewed study measured AZ gene expression under 3-CPA application, polar auxin transport flux in 3-CPA-treated tissue, TIR1/AFB receptor binding affinity for 3-CPA or its metabolites, or IDA-HAE pathway activation in any 3-CPA-treated system. The framework provides a map of the molecular landscape in which the 3-CPA mechanism question is situated, not a characterization of how 3-CPA could be involved. The research questions that would need to be addressed are identified in Section 8.

6.4. Limits of the Interpretive Context

Related-compound studies establish chemical and assay boundaries. CDA supplies a proposed explanation for fruit competition, while contemporary abscission-zone biology identifies possible molecular measurements. Neither framework establishes a CPA pathway. In particular, Bangerth [9] reports reduced auxin exports after thinning treatments, including CPA, but asks whether it is a cause or consequence; the cited doctoral original remains unavailable for inspection. The directly inspected experiments and these secondary accounts are therefore kept separate. Table 2 summarizes the crop evidence and its limits.

Table 2. Selected crop-response groups in the cited direct evidence. Years refer to publications, not continuous trial periods. Counts describe reports and can overlap between targets; they are not a total of independent experiments or all documents in the inventory. The 1960 pineapple structure–activity screen [49] is discussed separately in Section 4.1. Source-specific designs and limitations govern interpretation; no formal certainty rating is assigned.

Crop (Family)	Biological Target	Selected Cited Reports (Publication Years)	Evidence Level	Key Limitation
Prunus persica (Rosaceae) Peach	Fruitlet abscission/chemical thinning	22 local reports (1969–1990), including the 1970 experiments [14]; overlapping Cardinal campaigns [22,29,30]	Multiple programs with cultivar-, stage-, dose-, and formulation-dependent responses; thinning does not ensure commercial benefit	Historical cultivar base; no validation in current dominant cultivars. Physiological-status axis anchored chiefly by one bulletin [32].
Ananas comosus (Bromeliaceae) Pineapple	NDF flowering inhibition	8 reports; 1993–2026 [41–48]	Several programs and cultivars; protocol-dependent responses and incomplete formulation information	Different products, delivery, cultivars and designs; recent publication does not validate a common protocol or demonstrate mechanism [6,7].
Ananas comosus (Bromeliaceae) Pineapple	Fruit growth, maturation and related storage quality	5 growth/maturation reports; 1981–2025 [50–54]; 2 related storage reports; 2006–2007 [55,56]	Different preharvest and storage endpoints; cultivar- and protocol-dependent observations	Possible overlap between Soler reports and between Othman reports; reports are not independent trials. No common maturation or storage effect is inferred.
Ananas comosus (Bromeliaceae) Pineapple	Crown-size reduction	5 sources; 1978–2025	Reported in three countries and cultivars; formulations, designs and outcomes differ	Dalldorf [13] used a mixed formulation, did not describe replication, and recorded crown injury. Vieira [57] found no fruit-weight effect under drought; Imchen [58] used methanol carrier.
Ananas comosus (Bromeliaceae) Pineapple	Hormonal markers (ethylene tension)	2 reports; 1995 and 1996; one CPA experiment	Direct ethylene-pathway measurements alongside delayed flowering and tissue injury; causal mechanism unresolved	Same experiment in thesis and article; pooled tissues and injury limit inference; ethylene measurements also exist in peach [5]

Table 2. *Cont.*

Crop (Family)	Biological Target	Selected Cited Reports (Publication Years)	Evidence Level	Key Limitation
Vitis vinifera (Vitaceae) Grape	Berry-set reduction (bloom window)	1 source 1971	Preliminary cluster-dip screening; six clusters per treatment; ANOVA and Duncan comparisons	Cultivar- and stage-specific response with injury; not a whole-vine production protocol or measured mechanism
Carya illinoensis (Juglandaceae) Pecan	Nut abscission	4 reports; 1965–1977	Preliminary evidence from two theses and two proceedings reports; heterogeneous designs and identity information	Uncertain formulations; injury and incomplete statistical/harvest evidence; three reports from the Oklahoma program

6.5. Comparisons with Other Thinning Agents

Table 3 presents selected within-experiment comparisons from two historical reports in which CPA and another agent were included within the same design. Thinning, harvested fruit number, yield, and injury are considered together. These contrasts differ in cultivar, year, dose, and formulation; they do not establish an intrinsic potency ranking or a common effective concentration. Other reports using CGA 15281 or Ethrel without a CPA arm cannot supply a direct comparison. Cobianchi and Rivalta [14] also tested Ethrel, but in trials with their own hand-thinned controls; those treatment means do not provide a matched CPA-ethephon contrast and are not added to Table 3.

Table 3. Historical comparisons of CPA with other thinning agents in peach and nectarine. Reported doses are retained without conversion between chemical forms.

Trial and Crops	Treatments and Design	Reported Contrast	Injury and Limits
Cobianchi 1972 [35] Peach, Fortuna and Halford 1 1971	Fruitone CPA (7.9% amide + 0.4% acid), reported 200 ppm; ethephon 30/60 ppm. Five trees per treatment; hand-thinned reference, no untreated arm. Dose basis not separately specified.	Fortuna example: fruit drop 42.43% with CPA, 66.93/74.03% with ethephon, 51.20% by hand. Yield 173.1 versus 123.8/102.2 and 167.3 kg/tree, respectively. Ethephon differed from CPA for drop and yield; CPA and hand yield shared a group ($p = 0.05$). Table 2, p. 661.	Mild leaf injury with both agents (p. 662). More drop with ethephon accompanied lower yield in this trial. One year and a mixed CPA product; no inference about current safety or cost.
Retamales et al. 1990 [37] Nectarine, Early/Late Le Grand 1983/84–1984/85	CPA propionamide 200 ppm; CGA 250 ppm in the first season and 200 ppm in the second. Six whole trees per treatment; untreated and hand-thinned controls; seed about 12 mm.	Early Le Grand, 1983/84: harvested fruit/tree 388b with CPA, 222c with CGA and 530a untreated. Both chemicals differed from control and from each other. Fruit mass 113.4ab versus 123.7a g did not differ. Table III, p. 644; letters apply within cultivar/season/outcome.	Leaf fall in 1984/85 was descriptively greater with CPA: Early 6.77 versus 1.17 m^2/tree (Table II, p. 643; no test reported). Unequal first-season doses and reuse of Early trees constrain generalization; fruit-size and thinning responses varied.

The inspected corpus does not support a matched CPA comparison with NAA, 6-benzyladenine (6-BA), or ABA, or a ranking of costs, storage stability, labour requirements, or current human/environmental safety. Historical spray volumes and observed phytotoxicity are not substitutes for those assessments; Section 2.6 defines the safety and regulatory limits.

7. Physiological Hypotheses and Unresolved Mechanisms

The directly inspected CPA experiments establish developmental responses and selected physiological observations without establishing a causal pathway. Observations include ethylene evolution in peach fruit [5], bioassayed inhibitor- and promoter-like activity in abscising peach tissues [31], radiolabel distribution [16], and ethylene-pathway measurements alongside injury and delayed flowering in pineapple [6,7]. These assays measure different processes. CDA, altered auxin transport, embryo abortion, and particular receptor or abscission-zone pathways remain proposed explanations or research targets. Sections 7.1–7.3 assess their evidentiary limits.

7.1. Correlatively Driven Abscission as an Interpretive Hypothesis

CDA [9] is used here as a testable explanation for differences among fruitlets, as outlined in Section 6.2. A CPA-specific evaluation would need to distinguish altered endogenous IAA export from the movement of an applied labelled regulator and establish whether any transport change precedes and contributes to abscission. Physiological association or agreement with a model does not establish that causal sequence.

Bangerth developed CDA primarily from *Malus* × *domestica* (apple) data and explicitly noted that extension to other fruit species would require further experimentation, a caveat that applies directly to its use for *Prunus persica* and to any non-Rosaceae.

The proposed relevance of CDA to CPA remains interpretive. Bangerth [9] discusses auxin export in a secondary account, but the underlying doctoral experiment was unavailable for direct inspection and is not used as a verified transport result here.

The distinction between pool measurements and transport flux is consequential here. Extractable hormone pool concentrations report the net balance of biosynthesis, conjugation, and degradation at the time of tissue harvest; they do not establish the rate or directionality of polar, carrier-mediated IAA movement between cells, which is the process that CDA requires. No direct transport-flux evidence sufficient to establish CDA as the mechanism of CPA was identified in the inspected originals. The distinction limits causal inference from hormone-pool data and does not justify treating the unavailable doctoral source as if its methods had been examined [9].

Developmental-stage dependence and the field observations of variable thinning with the tree condition [32,33] provide reasons to test fruitlet competition as a possible interpretive framework. These observations do not establish a direction of crop-load effects or quantify carbohydrate availability, reserve status, or IAA export. Martin and Nelson [16] detected radioactivity near the ovule micropyle after radiolabelled CPA treatment, with slow, localized movement. Their experiment did not establish intact-compound accumulation at a 7–8 mm ovule stage or measure inter-fruitlet polar IAA transport. Neither line of evidence demonstrates CDA. Einset et al. [17] compared transport and metabolism of radiolabelled IAA and 2,4-D in excised *Citrus* pistils; that compound- and assay-specific experiment does not test CPA, inter-fruitlet competition, or CDA.

7.2. Pineapple Ethylene Measurements and Their Interpretive Limits

The pineapple measurements concern ethylene production, ACC oxidase activity, and MACC in stem tissue after CPA treatment [6,7]. They are more extensive than the ethylene

measurements in treated peach fruit [5], but they do not make pineapple the only crop with direct hormonal observations. The thesis and article report one CPA experiment. Elevated markers occurred in a treatment with tissue injury, and the article extended flowering observation from the thesis cut-off to delayed inflorescence appearance in all eight monitored plants.

Comparing these results with the flowering-inducing use of ethephon raises questions but does not isolate a causal ethylene pathway. The CPA experiment did not separate injury, ethylene production, downstream signalling, and delayed differentiation through targeted mechanistic interventions. The authors regarded the mechanism as unresolved [6,7]. Pool concentrations and pathway indicators should consequently be described as observations under the tested treatment conditions, rather than a directional signal that predicts opposite developmental outcomes for two compounds.

7.3. Contemporary Molecular Context

The molecular biology of the abscission zone has advanced substantially since the primary 3-CPA field-trial era. Tranbarger and Tadeo [10] review in detail the molecular events at the abscission zone during fruit separation including transcription-factor networks, cell-wall remodeling cascades, and hormone-signaling interactions involved in AZ activation, thus providing a contemporary molecular vocabulary that did not exist when the primary 3-CPA literature was generated. This framework, however, does not address 3-CPA-specific measurements.

No reviewed study has characterized the effects of 3-CPA on abscission-zone transcript levels, cell-wall enzyme activities, or transcription-factor networks in any crop system; the contemporary AZ framework therefore provides a descriptive backdrop for future 3-CPA mechanistic work, not a characterization of current 3-CPA mechanism [10]. TIR1/AFB-mediated auxin receptor signaling, through which auxin perception triggers transcriptional changes relevant to AZ development, has not been tested for 3-CPA in any reviewed source; no binding-affinity, downstream-signaling, or genetic evidence linking 3-CPA to TIR1/AFB pathway components appears in the reviewed literature.

The historical plum study [20] measured bioassayed growth-promoting activity after ethephon, whereas the peach [5] and pineapple [6,7] CPA studies measured ethylene-related variables. The assays, compounds, organs, and developmental endpoints differ. Together, they motivate questions about auxin–ethylene interactions, but they do not demonstrate a common pathway or a CPA effect on auxin transport. Whether such interactions cause any of the CPA responses reviewed here remains unresolved.

8. Evidence Limitations, Modern Relevance, and Future Research

8.1. Historical Evidence and Modern Validation

The *Prunus persica* evidence reviewed in Section 3 derives from a specific historical window, primarily the mid-1960s to mid-1970s, with later nectarine trials in the 1980s [37]. The studies were conducted under the orchard conditions, pest management, and germplasm of their time; the findings must be interpreted within their reported designs and conditions and do not establish efficacy in later cultivar bases or production systems. Geographic coverage remains limited and uneven across the inspected programs, even after the addition of reports from other countries. The inspected reports do not establish thinning efficacy in the contemporary cultivars and production systems that now define major peach-producing regions. The four response-conditioning variables documented in Section 3, developmental timing, cultivar identity, dose, and physiological status, were each assembled from separate experiments, and their interactions are inferential rather than directly quantified.

Extending the peach record to modern conditions would require, at minimum, controlled trials in commercially dominant post-1980 *Prunus persica* cultivars under contemporary orchard management, evaluating whether the developmental-staging and dose relationships in the historical record hold for modern germplasm. A factorial design that simultaneously varies developmental timing, cultivar identity, and dose within a single experiment would be the most direct way to estimate the interaction effects that cross-trial comparison can only suggest.

The physiological-status interpretation in Section 3.3 remains particularly tentative. Bowen and Powell's bulletin [32] includes replicated dose comparisons, but crop load and vigor were not experimentally assigned; Beutel et al. [33] add a qualitative observation rather than a controlled replication. Theses and institutional reports contribute original observations, but incomplete formulation identification, unreplicated comparisons, sector-based sampling, and missing harvest outcomes limit particular inferences, as detailed in Sections 3.3 and 5.2. Publication in a journal does not remove design limitations or create independent replication: Min's thesis [6] and the later article [7] describe the same CPA experiment, with different follow-up durations. The reliability of each inference therefore depends on its original methods and reporting.

Grape evidence in the inspected corpus remains limited to the 1971 report by Weaver and Pool [60], with six dipped clusters per treatment and a screening design rather than a validated whole-vine protocol. Pecan evidence includes four reports published from 1965–1977 [21,61–63], with different timing, design, and identity limitations; the three Oklahoma reports are not independent multi-institutional replications. Amling and Dozier add an Auburn source, but its one-page account lacks sample-size and statistical detail. These limits justify further evaluation, not the assertion that Burke is the sole source. Plum remains outside the main four-crop scope and requires its own compound- and formulation-specific assessment.

8.2. Mechanistic Gaps and Experimental Priorities

Three mechanistic questions remain unresolved by the original CPA experiments inspected here. These boundaries describe the consulted evidence rather than the absence of experiments elsewhere.

The inspected original CPA experiments do not establish a causal role for altered endogenous polar IAA transport. Extractable hormone activity, tissue hormone pools, and distribution of an applied radiolabel are distinct measurements [9,16,31]. Directional IAA transport assays combined with time-resolved growth and abscission observations would be needed to evaluate the proposed transport sequence. The secondary report of reduced exports [9] remains subject to verification of its doctoral source and to the cause-versus-consequence limitation stated by Bangerth.

The inspected original CPA experiments provide no molecular characterization of treated abscission-zone tissue. Transcriptomic or proteomic profiling in CPA-treated *Prunus persica* could test whether the pathways discussed in the contemporary framework [10] are involved without assuming that those pathways explain the historical responses.

The inspected sources provide no direct test linking CPA or an identified metabolite to TIR1/AFB receptor activity. Binding, signalling, or genetic experiments would be needed to evaluate that proposed connection; classification as an auxinic regulator alone does not establish it.

A further limitation is access to original mechanistic evidence underlying secondary accounts. The unavailable doctoral source cited by Bangerth [9] was not treated as a directly inspected study. Its absence from the local corpus does not establish that experimental data

are nonexistent or unavailable elsewhere. Any later recovery should be evaluated at the source level before it changes the mechanistic interpretation.

The coexistence of elevated ethylene-related markers, delayed flowering, and injury in the pineapple CPA experiment [6,7] remains unresolved mechanistically. Future work should distinguish tissue injury and ethylene production from pathway-specific signalling and floral-development timing while retaining the chemical identity and delivery conditions of the applied material.

Each of the directions outlined above addresses a defined gap; none constitutes a recommendation for current use of 3-CPA in any crop system.

9. Conclusions

The inspected reports document several developmental responses to CPA in four selected crop systems, with uneven publication coverage: 1969–1990 in peach/nectarine, 1960–2026 in pineapple, 1971 in grape, and 1965–1977 in pecan. The overall span does not establish 6 decades of evidence in each crop. The breadth and credibility of that evidence vary with formulation identity, cultivar, experimental design, measurement, statistical reporting, and access to originals. Repeated reports of a campaign, descriptive progress reports, and secondary mechanistic accounts do not provide interchangeable support. The review therefore identifies bounded observations and unresolved questions rather than a validated mechanism or a contemporary production protocol.

Further work should use chemically identified materials and controlled designs to distinguish developmental response from injury, compare contemporary cultivars where justified, and measure proposed causal processes directly. Directional IAA transport, hormone signalling, and floral differentiation require different assays; an association among their indicators does not establish a shared pathway. Historical evidence, including the expanded but still preliminary pecan record, provides questions for such work. It does not establish current efficacy, safety, or authorization of a commercial formulation.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/agriculture16212399/s1; Table S1: Peach and Nectarine Responses to 3 CPA. Concentration basis is retained as reported; NR means no inferential test was established for the displayed contrast.

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