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*Review*

# Progress in Fruit Cracking Control of Gibberellic Acid and Abscisic Acid

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**Abstract:** Fruit cracking or splitting is a severe physiological disease that significantly affects appearance and quality, compromising the commodity value of fruit and causing substantial economic losses to the producers of several fleshy fruit crops. The growth-promoting plant hormone gibberellins (GAs) and growth-inhibiting abscisic acid (ABA) antagonistically regulate numerous processes throughout the plant life cycle. The homeostasis of GA and ABA plays a significant role in the normal growth and development of fruits, and the imbalance of them may lead to the occurrence of cracking or splitting during the process of fruit growth, development, ripening and postharvest storage. The pathways of GA and ABA metabolism and signaling have been studied widely, and the major components are well characterized, including the genes encoding major biosynthesis and catabolism enzymes and the key signaling components. Nevertheless, our knowledge of the mechanisms of GA and ABA governing fruit cracking is not comprehensive enough. In this review, we summarize the advances in understanding the effects of endogenous GAs and ABA contents in fruits and exogenous GAs and ABA treatments on fruit cracking, and we endeavor to provide some genetic cues on the function of GAs and ABA responsible for fruit cracking modulation. The progress in understanding the molecular bases underlying the actions of GAs and ABA in fruit cracking coordination control will facilitate breeding strategies of cracking-resistant ideotypes of fruits, and also carry great theoretical significance in guiding the establishment of integrated prevention and control measures in fruit cracking.

**Keywords:** progress; plant hormones; gibberellins; abscisic acid; metabolism genes; fruit cracking modulation

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## 1. Introduction

Cracking or splitting, a common symptom of fruit surface fractures encountered in many fruit crops, has been described as "the physical failure of the fruit skin". This occurs when the internal growth of fruits is not in harmony with external environmental factors [1–3]. As a common and severe physiological disorder, cracking has an adverse and common bearing on both natural beauty and yield performance. And cracking reduces the fruit marketability as it causes unfavorable effects in quality, such as poor appearance and severe nutrient loss, decreased shelf life and increased infections by certain fungi or bacteria, and so on, resulting in unquantifiable losses in the fresh market. Thus, the cracked or splitted fruits can only be used in processing industries before they are infected by fungi [4–9]. In spite of fruit cracking having been studied for decades, and many fruit breeders are also working

hard to incorporate crack resistance into these fruit crops to enhance their crack resistance, very slight advances have been made in understanding the physiology and genetics of fruit cracking. In turn, this also makes it particularly difficult to recommend effective measures to prevent fruit from cracking. Therefore, it is significant to better investigate and understand the mechanism of cracking in different fruits to reasonably control this physiological disorder.

Fruit cracking or splitting has challenged the scientific community for decades; the first systematic study and reports about sweet cherry (*Prunus avium* L.) cracking began more than 90 years ago [10], and since then, a large number of studies with the fundamental aim to classify the CR (cracking-resistant) and the CS (cracking-susceptible) sweet cherry cultivars based on their susceptibility to cracking followed [11–13]. Furthermore, researches have reported that many crops, such as litchi (*Litchi chinensis* Sonn.) [14,15], tomato (*Lycopersicon esculentum* Mill.) [16], persimmon (*Diospyros kaki* Thunb.) [17], peach (*Prunus Persica* L.) [18,19], grape (*Vitis vinifera* L.) [20], apple (*Malus domestica*) [21,22], sweet orange (*Citrus sinensis*) [23], pomegranate (*Punica granatum* L.) [24], fresh fig (*Ficus carica* L.) [25], pear (*Pyrus* spp.) [26,27], jujube (*Ziziphus jujuba* Mill.) [28], watermelon (*Citrullus lanatus*) [29] and strawberry (*Fragaria* × *ananassa* Duch.) [30], are liable to crack or split (Figure 1), and the cracking rate is about 30%, in some varieties as high as 60%–80%, in serious years up to 90% or more, and the loss caused by cracking reaches 30%~60% or even more, causing considerable economic loss and agricultural resource waste [5,6,8,31].

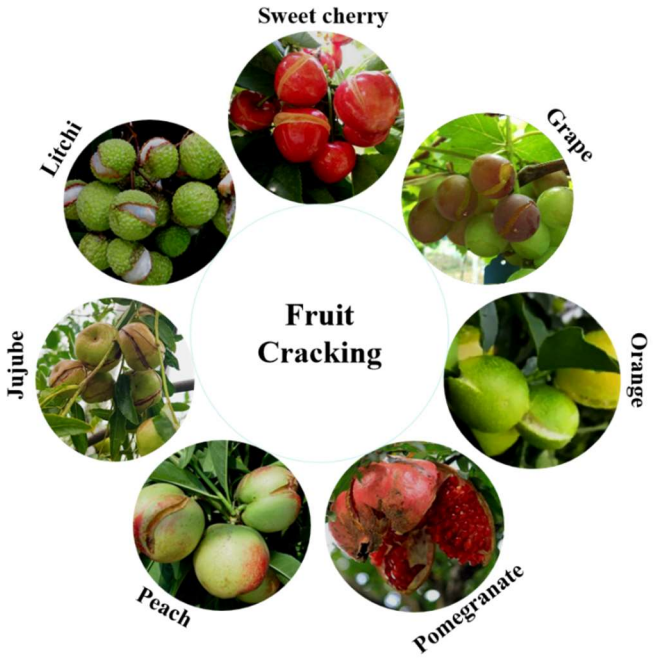


**Figure 1.** Cracking in several different fruit species. Seven common fruits that are liable to crack, including sweet cherry, grape, orange, pomegranate, peach, jujube and litchi, are represented. Among them, sweet cherry (https://baijiahao.baidu.com/s?id=1668282007129830555&wfr=spider&for=pc, accessed on 1 June 2020), grape (https://baijiahao.baidu.com/s?id=1607020440803990504, accessed on 26 July 2018), orange (https://www.sohu.com/a/381112523_99993524, accessed on 18 March 2020), pomegranate (https://www.cnhnb.com/xt/article-1274.html, accessed on 19 September 2021), peach (https://www.sohu.com/picture/397690934, accessed on 26 May 2020) and litchi (https://v.youku.com/v_show/id_XMjgzNjI1NDY1Ng==.html?, accessed on 20 June 2017).

It is well known that the underlying mechanisms of cracking are comprehensive and quite complex. It has been reported that the high occurrence of fruit cracking or splitting can be influenced by several factors, such as genetic characteristics, stressful environment cues, orchard management conditions, fruit growth rate, postharvest storage

factors, physiological, biochemical, anatomical and plant hormones [6,31–33]. Noteworthily, under the same external environmental conditions, including light, temperature, wind and rainfall; and the same orchard management conditions, including light, nutrition, irrigation, minerals and growth regulators, different fruits species or cultivars show differences in cracking susceptibility. Several factors influencing the susceptibility to fruit cracking have been observed, related to fruit traits such as size, shape, hardness, growth rate, water content and peel characteristics. These characteristics include the anatomy and strength of the fruit skin, stomata on the fruit skin, cuticular properties, osmotic concentration and the cuticle [2,34–36]. Additionally, the water capacity of the fruit pulp and the expression of genes related to the growth stage of the fruits are significant. In addition, the regulation of phytohormone and plant growth regulators (PGRs) are very vital factors of cracking due to the growth and development of fruits cannot be separated from the regulation of phytohormones. The homeostasis of endogenous phytohormones plays a vital role in the normal growth of the pericarp, and its imbalance may cause fruit cracking or splitting [37–40]. PGRs which have similar functions to the endogenous plant hormone are well known and also have a certain effect on fruit cracking. Although foliar sprays with PGRs could be an important orchard management method, little is known about the effectiveness or the response of fruits to PGRs, and the molecular details and physiological roles of each of them in mediating cracking are vagued.

Remarkably, during the past decades, the functions of phytohormones in fruit cracking determination have been unraveled mainly through investigating the effect of endogenous phytohormones [ABA, GAs, IAA (3-Indoleacetic acid), trans-zeatin-riboside, trans-zeatin, isopentenyladenine, brassinolides (BRs) and JA (jasmonic acid)] content in the pulp or pericarp of fruits and exogenous PGRs treatment, the commonly used PGRs which are GA3, GA4+7, ABA, BRs, naphthalene acetic acid (NAA), cytokinin (CTK) and JA, and so on, and the expression levels of the genes that are involved in phytohormone metabolic pathways [38–45]. Among them, the growth-promoting GAs and growth-inhibiting ABA antagonistically regulate many developmental processes and also play pivotal roles in modulating fruit cracking during fruit growth, development, ripening and postharvest storage. Previous studies have shown that the normal (uncracked) fruits contain higher level of GAs and lower ABA, and an imbalance between the two phytohormones of GA and ABA in fruit peels can lead to cracking in different fruit crops [1,38,46]. Here, progress in ABA and GAs that regulate fruit cracking is reviewed to provide the basics for controlling fruit cracking. As such, understanding the underlying function mechanism of antagonistic ABA and GAs mediating fruit cracking would be better for governing the cracking or splitting of different fruit crops properly.

## 2. Study Material and Methods

The study focus on some fruit species, including litchi, sweet cherry, jujube, pomegranate, mandarin, lemon, pear and apple. This review mainly summarizes the research progress on the effects of endogenous GAs and ABA contents in fruits, the function of the exogenous GAs and ABA treatments in fruit cracking control from 1969 to 2023, and the potential genes related to GAs and ABA metabolic pathways and possibly involved in regulation of fruit cracking. During the process of preparing this review, several search engines were scanned, mainly including Web of Science (https://www.webofscience.com/wos/woscc/basic-search, accessed on 9 February 2024), PubMed (https://pubmed.ncbi.nlm.nih.gov/, accessed on 9 February 2024) and CNKI (https://www.cnki.net/, accessed on 9 February 2024).

## 3. Progress in Biological Function of GA and ABA

### *3.1. Biological Function of GA*

GA is diterpenoid phytohormone that has multiple biological functions acting throughout the plant life cycle [47]. As a growth-promoting plant hormone, GA is crucial for many aspects of plant growth and development, including influencing seed germination, stem elongation, flower induction, anther development and seed and pericarp growth, especially

in promoting cell elongation [47–51]. Meanwhile, GA can also promote plant root growth, preserve flowers and fruit, promote peel development, increase fruit weight and increase yield. These effects also grant GA the ability to reduce the fruit cracking rate through a specific mechanism that primarily:: delays fruit maturity; enhances peel elasticity; increases the deposition of stratum corneum components to boost the elasticity of the stratum corneum; reduces the activity of PME (Pectin methylesterase) and PG (Polygalacturonase) to postpone the softening of the fruit; enhances the plasticity of the cell wall; and maintains the fruit firmness [52–56]. Notably, it is well known that GA is widely used in many horticultural crops to improve fruit set, increase fruit size and firmness [26,57–60], and make pomegranate [24,61], litchi [14] and sweet cherry [62] fruit more resistant to cracking. And some studies have suggested that the foliar spraying of PGRs that function similar to active GA could help to improve quality and reduce the occurrence of cracking, and the application of exogenous GA can reduce fruit cracking because of the increased GA levels in fruits [15]. However, to our knowledge, evidence that GA can decrease the cracking or splitting of fruit is contradictory. Altogether, it is important to further determine whether GA can enhance fruit crack resistance well.

### *3.2. Biological Function of ABA*

ABA is a major growth-inhibiting phytohormone with a sesquiterpene structure, which is common and widely existing in many plant species, regulating a broad range of plant traits, and is especially significant for adaptation to adverse environmental conditions [63]. Studies have shown that ABA plays important roles in regulating various processes of the plant growth and development, and it can significantly inhibit the growth and development of most plant crops through promoting the rapid senescence and fall off of flowers, leaves, fruits and other organs of most plants, and ABA can also stop the growth of seeds and induce the seeds to quickly enter a dormant state especially [64,65]. It is well known that ABA is involved in fruit cuticle integrity, controls the response of plants to environmental stress, and also plays significant roles in the process of fruit adaptation to abiotic stresses and fruit cracking control. Changed levels of ABA have been related to cracking or splitting in many fruit crops. For example, the accumulation of ABA in aril is necessary for the growth and development of aril, but a higher content of ABA could induce fruit cracking in litchi [39]. In addition, several research studies have reported that ABA affects fruit development probably by closing leaf stomata, reducing water loss, and promoting water inflow into the fruit, thereby increasing internal fruit pressure, and thus resulting in an increased cracking rate of tomato fruit [42,66].

## 4. Progress of GA and ABA in Fruit Cracking Control

### *4.1. GA in Fruit Cracking Control*

#### 4.1.1. Effect of Endogenous GA Content on Fruit Cracking

Changed levels of endogenous GA have long been associated with cracking in many fruit species [28,41,67,68]. As early as 1986, Sharma and Dhillon investigated the relationship between the endogenous GA level and cracking of litchi fruit and observed that GA content in the seed and pericarp of litchi is higher in cracked fruits than in non-cracked 'Dehradun' litchi [14]. Then, through comparing the content difference of endogenous plant hormones in cracked and uncracked jujube fruits, Cao et al. (2014) found that the content of active $GA_3$ in the exocarp of the CS cultivars of jujube was significantly higher than those of the CR jujube varieties [41]. Analogously, the research of Wang et al. (2020) in jujube cracking further reported that the $GA_3$ content was significantly higher in the cracked parts than the uncracked parts of the CS cultivar 'Fucuimi' jujube [69]. Based on these findings, it is speculated that there might be a positive relationship between fruit cracking and the GA level. Inversely, it was shown that the endogenous GAs level in cracked fruit was significantly lower than that in normal jujube fruit [70]. In addition, it was also observed that the cracking index was negatively correlated with the concentration of endogenous GA in research studies of jujube cracking [28]. Nevertheless, Yilmaz and Ozguven (2006)

estimated the contents of exogenous phytohormone in the fruit peel of pomegranate cultivars, while no significant differences were detected between the $GA_3$ level in cracked and uncracked pomegranate fruit peels [38]. Taken altogether, these above results suggest that the content of endogenous GA in the fruits is closely related to the cracking of several fruit species, but the effect of endogenic GAs on cracking might exhibit some differences in different species or among different cultivars of the same fruit species, while the influence differences of GAs among fruits cultivars have not been clearly identified, which will need further study.

#### 4.1.2. Effect of Exogenous GA Treatment on Fruit Cracking

Many researchers have investigated the roles of the phytohormone GA in the cracking or splitting of fruit by using exogenous GA analog treatments (Table 1). For instance, it has been suggested that exogenous $GA_3$ can reduce the cracking rate of litchi [71], and the ratio of cracked 'Dehradun' litchi was effectively reduced by spraying with $GA_3$ compared with those of the control [14,15]. Notably, the application of $GA_3$ (20 mg /L) had no significant effect on cracking, while treating with a mixture of $GA_3$ (20 mg /L) and 2,4-D (20 mg /L) could significantly reduce the fruit cracking of 'Nova' mandarin [23,72]. Josan et al. (1998) reported that $GA_3$ treatments could increase the contents of IAA and GAs but reduced the ABA levels in the peel and pulp of 'Baramasi' lemon fruits and reduced the ratio of cracked fruit compared to the control [73]. And Maotani et al. (1990) reported that GA tapes containing 6% GA ($GA_3$:$GA_4$ = 9:1) tied at the calyx ends or peduncles at about 30 DAFB could reduce fruit cracking of the 'Kosui' and 'Niitaka' Japanese pear fruit [74].

**Table 1.** Fruit cracking control of exogenous GAs treatment in different fruit species.

| Species | Variety | PGRs Treatment | Treatment Methods | Cracking | References |
|---|---|---|---|---|---|
| Litchi | 'Dehradun' | 50, 75, 100 ppm $GA_3$ | 2-year-old trees; applied at 7-,11-, or 15 day intervals from the early stages of development until harvest | Decreased | [75] |
| | 'Dehradun' | 25 and 50 ppm $GA_3$ | four sprays (at fruit set + 2 weeks later + 4 weeks later + 6 weeks later) | Decreased | [15] |
| Jujube | 'Lizao' | 15 mg/L $GA_3$ | sprays at 3 and 2 weeks before the commercial harvest date | Decreased | [28] |
| | 'Fucuimi' | 15 mg/L $GA_3$ | six foliar sprays began at 7 DAFB; and at 10 days once | Decreased | [76] |
| Cherry | 'Justyna', 'Tamara', 'Regina' | 10% $GA_3$ (800 L/ha) | 10–11-year-old trees; applied once 10–12 days or 15–20 days before harvest with a tractor sprayer | Decreased 9–11% | [67] |
| | 'Binga', 'Sam' | 10 or 40 ppm $GA_3$ | pre-harvest single or repeated foliar spray | Increased | [62] |
| | 'Merton Premier', 'Bing', 'Dawson' 'Sweetheart' | 10, 20 or 30 ppm $GA_3$ | single or multiple treatment | Ns | [77,78] |
| Pomegranate | 'Hicaz', 'Silifke Aşısı' | 100, 150, 200 mg/L $GA_3$ | 5-year-old trees; applied in the second week of August and September | Decreased | [79] |
| | 'Manfalouty' | 80 ppm $GA_3$ | pre-harvest sprays | Decreased | [24] |
| | 'Wonderful' | 75 or 150 mg /L $GA_3$ | foliar spray, in July | Decreased | [80] |
| Mandarin | 'Nova' | 20 mg /L ($GA_3$ + 2,4-D) | applied once or twice after June drop | Decreased | [23] |
| | 'Nova' | 20 mg /L $GA_3$<br>20 mg /L ($GA_3$ + 2,4-D) | 7–10-year-old trees; foliar sprays (5–7 L/tree); treat twice at 60 and 30 d before splitting | Ns<br>Decreased | [72] |
| Lemon | 'Baramasi' | 10 or 20 ppm $GA_3$ | 10-year-old tree; sprays on 15 and 30 May DAFB | Decreased | [73] |
| Pear | 'Kosui', 'Niitaka' | GA tapes contains 6% GA ($GA_3$:$GA_4$ = 9:1) | tied at the calyx ends or peduncles at about 30 DAFB | Decreased | [74] |
| Apple | 'Pink Lady' | 20 mg/L ($GA_{4+7}$ + BA) | treated at 50–65 DAFB | Decreased 20.6% | [68] |

Ns: No significant difference between the treatments and the controls. DAFB: Days after full blossom.

In recent years, exogenous $GA_3$ has consistently been used for controlling fruit cracking in various horticultural crops, and studies have shown that exogenous GAs treatment was available for reducing the fruit cracking rate of several fruit crops (Table 1). For example, in the research studies of the cracking control of jujube fruits, it was observed that exogenous $GA_3$ (15 mg/L) treatment at 3 and 2 weeks before the commercial harvest stage lowered the fruit cracking or splitting rate of 'Lizao' [28]. It was also observed that cracking of 'Fucuimi' fruit that underwent spraying treatment with $GA_3$ (15 mg/L) was also lowered compared to the control [2]. Similarly, exogenous $GA_3$ treatment decreased the rain-induced cracking rate of the 'Justyna', 'Tamara' and 'Regina' cherry varieties by 9%–11% [67]. Spraying a mixture of $GA_3$ (40 ppm), calcium hydroxide, zinc sulfate and boron (50 ppm) minimized the incidence of cracking on pomegranate young fruits [38]. Meanwhile, $GA_3$ treatment also caused a reduction in the splitting of the pomegranate varieties 'Hicaz', 'Silifke Aşısı' [38], 'Manfalouty' [24]. More recently, a further 2-year study of pomegranate fruit cracking control also uncovered that foliar spraying with exogenous $GA_3$ (75 or 150 mg /L) in July could substantially decrease the cracking rate [79]. Multiple treatments of 'Pink Lady' apple fruit with $GA_{4+7}$ and BA at early phenological stages (50–65 DAFB, days after full blossom) results in an increased epidermal cell density and reduced calyx-end cracking disorder until fruit mature and harvest (210 DAFB), implying exogenous PGRs have a long-term effect of the treated plants [68,81]. On the contrary, $GA_3$ sprays increased the cracking of cherry [57]; Cline and Trought (2007) also demonstrated that pre-harvest single or repeated foliar applications of $GA_3$ (10 or 40 ppm) in the CR variety 'Binga' and the CS variety 'Sam', increased both fruit firmness and fruit cracking in sweet cherry [62], while several studies showed that single or multiple $GA_3$ treatment on the cultivars 'Merton Premier', 'Bing', 'Dawson' and 'Sweetheart' had no influence on the cracking of sweet cherry [77,78]. Interestingly, Agusti et al. (2002) found that the $GA_3$ influence on fruit cracking might be based on the application time: the application of $GA_3$ at flowering increased the cracking of citrus fruits, but decreased fruit cracking after the end of the June drop slightly [23]. Given the above findings, it is suggested that exogenous GAs application is closely related to cracking or splitting control and likely contributes to restrain cracking of most fruit species effectively, while the effect of exogenous GAs on fruit cracking also might be related to the species, variety of fruit and the application time of exogenous GAs. It is important to further clarify the molecular mechanism of exogenous GAs' regulation on fruit cracking and explore the potential related genes involved in the GA metabolism pathway and fruit cracking control.

#### 4.1.3. GA Metabolism Pathway Genes in Fruit Cracking Control

It is well known that both the endogenous GAs level and exogenous GAs treatment play important roles in fruit cracking control. Hadjipieri et al. (2021) showed that exogenous plant hormones can change the morphology of the epidermis and cuticle, which then improves the physical properties of the fruit exocarp and thus decreases fruit cracking or splitting and improves the control of the fruit quality overall [82]. And applications of exogenous $GA_3$ decreased cracking of fruits, likely because the treatment of $GA_3$ could increase the deposition of cuticular material in the epidermis and increase elasticity [34]. In recent years, to further clarify the molecular mechanism of GA regulation in fruit cracking, several potentially important genes related to fruit cracking and involved in GA metabolism pathway have been successfully investigated.

Previous research studies have studied the metabolism and signaling pathways of GA widely. In higher plants, the biosynthesis of active GA is catalyzed by six key enzymes, including CPS (*ent*-copalyl diphosphate synthase), KS (*ent*-kaurene synthase), KO (*ent*-kaurene oxidase), KAO (*ent*-kaurenoic acid oxidase), GA20ox (Gibberellin 20-oxidase) and GA3ox (Gibberellin 3-oxidase); the deactivation of active GAs is catalyzed by GA2ox (Gibberellin 2-oxidase) [47]; and the GA signaling pathway contains three crucial components, including the GID1 (GA insensitive dwarf 1) and the GID2/SLY1 (F-box protein), as well as the DELLA protein, a repressor of GA signaling [83]. GAs levels also regulate the

stability of the DELLA protein. The interaction between GA-GID1 and DELLA promotes the interaction with GID2/SLY1, promotes the formation of a triple complex GA-GID1-DELLA and then induces the degradation of DELLA [84]. In addition, the crosstalk of GA and other environmental and endogenous signals (light, temperature, BRs, auxin, ABA, etc.) is mainly dependent on the interaction between DELLA and BZR1 (Brassinazol resistant 1), PIFs (Phytochrome interacting factors), ARFs (Auxin responsive factors) and ABI3/5 (Abscisic acid insensitive 3/5) [51,84,85]. The functional diversity of GA is the direct consequence of DELLA protein activity [51]. The changes in the gene expression level of the GA pathway directly affect the active GA content and its biological function. For example, *GA2ox* negatively regulates the GA levels, and either upregulated *GA2ox7* or *GA2ox8* can reduce GA levels [86]. The GAs levels are positively correlated with the expression level of the *GID1* gene, and over-expressing of *GID1* could rescue the dwarf of the mutants *sly1* and *gid2* through altering GA content [87].

To identify candidate genes and further investigate the molecular mechanism of GA-controlled fruit cracking, RNA sequencing was first used for de novo assembly and characterization of the cracked and uncracked pericarp of litchi, and the expression levels of genes involved in GA metabolic and signaling pathways were analyzed [1]. Furthermore, it was found that five genes involved in the GA pathway, including two GA biosynthesis genes (*LcKSs*), two GA deactivation genes (*LcGA2oxs*) and one GA receptor gene (*LcGID1*), were differentially expressed genes (DEGs) in cracked fruits compared to uncracked litchi fruits (Table 2). Notably, of these DEGs, two *LcKS* genes and one *LcGID1* were monitored and found to be downregulated more than two-fold in peels of cracked fruits compared to the normal litchi fruits [1]. Conversely, two *LcGA2ox* genes, responsible for the deactivation of active GAs through 2$\beta$-hydroxylation, were upregulated more than two-fold in the peels of cracked fruits than that in normal litchi fruits [1]. These results further implied that GAs were highly accumulated in uncracked litchi fruits. Moreover, two GA-regulated protein genes (*GPRs*, *Lc.1.532* and *Lc.1.534*) (Table 2) related to GA were found to be downregulated in the pericarp of the CS cultivar 'Nuomici' litchi, resulting in differences in the mechanical strength of the pericarp and the development of fruit. Meanwhile, one GA receptor gene *LcGID1c* (*Lc.8.678*) (Table 2) was also downregulated in the aril of 'Nuomici' [88]. During the fruit development process of apples, *MdGID1b* (Table 2), which mediates GA perception in fruit ovules by interacting with DELLAs, is markedly upregulated, and *MdGID1b* is also significantly upregulated in the skin of mature apple fruits treated with the GA4+7 and BA compared with the control and causes increased epidermal cell density and prevents the cracking initiation of apple fruits [68]. Further, following the GA4+7 and BA treatment, the expression of *MdGID1b* was also significantly upregulated in the pericarp of mature apple fruit [68]. In jujube, Hou et al. (2022) found that the GA biosynthesis gene *ZjGA20ox1* (*gene19292*) and GA deactivation (*ZjGA2ox*, *gene243*) were gradually downregulated in the cracked and uncracked fruit samples of the CS variety 'Jinsixiaozao' and non-cracking CS variety 'Muzao' [45]. Taken altogether, these above findings suggested that the changed expression level of genes involved in GA pathways are closely related to fruit cracking, especially for *KS*, *GA20ox*, *GA2ox*, *GID1* and *GPRs* which are likely the main GA-pathway-related genes regulating fruit cracking, but the underlying molecular mechanisms of these genes regulating cracking need to be further studied.

**Table 2.** Potential cracking-related genes involved in GA pathways.

| Genes Annotation | Gene Name | Gene Accession | Species | References |
|---|---|---|---|---|
| *ent*-kaurene synthase | *LcKS* | *Unigene0009890*<br>*Unigene0009891* | Litchi | [1] |
| GA 20-oxidase | *ZjGA20ox* | *gene19292* | Jujube | [45] |
| GA 2-oxidase | *LcGA2oxs* | *Unigene0034731*<br>*Unigene0040846* | Litchi | [88] |
| | *ZjGA2ox* | *gene243* | Jujube | [45] |
| GA insensitive DWARF1 | *LcGID1* | *Unigene0002046* | Litchi | [1] |
| | *LcGID1c* | *Lc.8.678* | Litchi | [88] |
| | *MdGID1b* | *MDP0000929994* | Apple | [68] |
| GA-regulated proteins | *LcGPRs* | *Lc.1.532/Lc.1.534* | Litchi | [88] |

### *4.2. Abscisic Acid in Fruit Cracking Control*

#### 4.2.1. Effect of Endogenous ABA Content on Fruit Cracking

Previous studies indicated that the content of endogenous ABA is closely related to cracking in various fruits, and the higher ABA in the pericarp of fruit can easily induce fruit cracking or splitting. For example, Sharma and Dhillon (1998) reported that the ABA levels in the aril and peel of cracked fruit were higher than those of uncracked litchi fruits [89]. The ABA content and fruit cracking ratios were higher in control fruits in lemon compare to the $GA_3$ and NAA treatments [73]. The endogenous ABA concentration in the peels of cracked fruits was generally higher than that in the peels of uncracked pomegranate fruits [38]. In litchi, a balance between pericarp strength and aril expanding pressure can be related to litchi cracking, which can occur due an imbalance of plant hormone metabolism, and the content of ABA in the CS variety 'Baitangying' is higher compared with the CR variety 'Feizixiao' pericarps [44,88]. Marboh et al. (2017) observed that the accumulation of ABA in the aril is required for the development of the aril, but a higher ABA level can induce cracking of litchi fruit [39]. Moreover, fruit cracking or splitting in the 'Muzafarpur' litchi is directly related with higher ABA in the pericarps of the litchi fruits [6]. Romero and Lafuente (2020) reported that the deficiency of ABA can alter the metabolism level and morphology of the waxy layer, causing increased permeability of the cuticle during the progress of sweet orange fruit ripening, which might induce the occurrence of fruit cracking [90]. Meanwhile, in the study of ABA content in fruit cracking control of jujube, it was found that the high concentration of ABA promotes fruit senescence and accelerates the softening of pericarp tissue during the growth and ripening process of jujube fruit, resulting in fruit cracking, and the split index of jujube was positively correlated with the level of endogenous ABA. Yang et al. (2009) illustrated that the endogenous ABA level in cracked jujube was significantly higher than that in the corresponding part of normal fruit by comparing the difference of endogenous hormones in cracked and uncracked jujube fruits [70]. During the high occurrence stage of jujube cracking, it was found that the amount of ABA in the exocarp of a CS cultivar was higher than that of a CR cultivar [41]. And then, Wang et al. (2020) revealed that the endogenous ABA was high in the cracked parts of the jujube cultivar 'Fucuimi' [69]. Recently, Liu et al. (2023) reported that the endogenous ABA in the peels of CS jujube was remarkably higher than that in CR jujube individuals, and the ABA in the exocarp was higher than that in the mesocarp throughout the fruit development of jujube [91]. Based on these studies, it is suggested that fruit cracking may be positively correlated with the content of endogenous ABA, which is consistent with the results on the relationship between ABA and fruit cracking in litchi and pomegranate. Therefore, it is reasonable to speculate that ABA is one of the key factors that contributes to the occurrence of fruit cracking or splitting. On the contrary, in the study of grape, it was reported that the decreased ABA could reduce the enzyme activity of PME and PG and delay the degradation of pectin, increase the amount of exocarp

protopectin, enhance the mechanical properties of the exocarp and thus improve the fruit crack resistance of the grape berry [92]. In conclusion, all of these results implied that the level of the endogenous phytohormone ABA in fruits is closely related to the cracking or splitting of many fruit species, while the effect of endogenous ABA on fruit cracking might be different in different fruit species.

#### 4.2.2. Effect of Exogenous ABA Treatment on Fruit Cracking

Recently, to investigate the roles of phytohormones in fruit cracking, the application of exogenous plant growth regulators was usually carried out in fruit cracking control. The effect of exogenous ABA treatment on fruit cracking has also been extensively and successfully studied and verified, and it is observed that exogenous ABA treatment induces cracking or splitting in several fruit species. For example, the application of exogenous ABA through spraying treatment could increase the expansion rate and fruit cracking rate of litchi, jujube and tomato fruits [93]. Treating with exogenous ABA can increase water movement into the fruits and promote enlargement of the fruits, and treating with ABA also induces a tendency for the fruit to crack. Studies have shown that exogenous ABA could increase the rate of fruit cracking in jujube [76,91] and tomato fruits [42,66]. The research of Gutiérrez et al. (2021) indicated that treating with exogenous ABA before the fruits were harvested could increase cell wall and cuticle wax components at maturity and improve the crack resistance of sweet cherries, meaning that ABA-induced fruit cracking is mainly regulated by the cell wall metabolic pathway [94]. It has also been shown that spraying treatment with ABA (50 mg/L) had the best anti-cracking effect on 'Fucuimi', which decreased cracking by 39% compared with that of the control (Table 3) [76], and the fruit cracking index of 'Pingshunbenzao' treated with exogenous ABA solution (50 mg/L) at the white-ripening stage was significantly increased compared to the control that treated with sterile ultrapure water (Table 3), indicating that the ABA had a certain regulatory effect on jujube cracking [91]. At the same time, ABA was induced after water absorption in sweet cherry fruits, and the expression levels of the genes related to ethylene synthesis were increased, which was because ABA stimulated the production of ethylene in sweet cherry [95], and then the cell wall was degraded under the action of ethylene and finally the fruit was cracked [36]. Taken altogether, the above findings indicate that treating fruits with growth-regulating agents to modulate the plant growth and development cycle and reduce susceptibility to growth-induced cracking or splitting and the exogenous ABA can play either a dominant or supportive role in manipulating cracking or splitting in the development of several fruit species. Further, it is important to explore and clarify the molecular mechanism of the related genes involved in the ABA metabolism pathway and fruit cracking control.

**Table 3.** Fruit cracking control of exogenous ABA treatment in different fruit species.

| Species | Veriety | PGRs Treatment | Treatment Methods | Cracking | References |
|---|---|---|---|---|---|
| Jujube | 'Fucuimi' | 50 mg/L ABA | six foliar sprays began at 7 DAFB; once every 10 d | Decreased 39% | [76] |
| | 'Pingshunbenzao' | 50 mg/L ABA | 3 foliar sprays began at WR; once every 7 d | Increased | [91] |
| Cherry | 'Bing' | 0.1 mM ABA,<br>0.4 mM MeJA<br>0.1 ABA + 0.4 mM MeJA | single applications at 20 d DAFB or 60 DAFB (days after full blossom) | Decreased<br>Decreased<br>Decreased 87% | [96] |
| Tomato | 'Craigella' | 0.5 mg/L ABA | sprayed 1× per week for 3 weeks with a backpack applicator until the plants were completely covered with the solution | Increased 10.2% | [66] |

DAFB: Days after full blossom. WR: white ripening period (13 August).

4.2.3. ABA Metabolism Pathway Genes in Fruit Cracking Control

At different periods of fruit cracking, the contents of different forms of ABA in fruit were different, which is related to the biosynthesis, metabolism, transport and regulation of ABA. Many studies have suggested that changes in endogenous ABA levels and exogenous PGRs ABA treatment can play either a primary or auxiliary role in regulating fruit ripening and fruit cracking and influence fruit quality traits during fruit growth and development [42,91,93,94,97]; however, compared with the mechanism of ABA controlling fruit ripening, our knowledge of the underlying molecular cues of ABA mediating fruit cracking is still at the beginning stage. Studies have shown that ABA may induce fruit cracking mainly through regulating cell wall metabolism and then affect fruit yield, quality and economic benefits. Thus, elucidating the molecular mechanism of ABA-mediated fruit cracking has considerable potential to improve our understanding of both fruit cracking during ripening and to develop new fruit traits and varieties, especially the cracking-resistant fruit varieties.

Comprehensive knowledge of the key genes involved in ABA biosynthesis, metabolism, transport, signal transduction pathways and regulation of ABA revealed that the synthesis of active ABA is derived from zeaxanthin [98], and some enzymes such as ZEP (Zeaxanthin epoxidase), NCED (9-cis-epoxycarotenoid dioxygenase) and AO (aldehyde oxidase) may play key roles in regulating ABA biosynthesis in higher plants. Among them, ZEP can produce the precursor of ABA biosynthesis and convert zeaxanthin to violaxanthin [99,100], while the deactivation of active ABA is catalyzed by key enzymes: ABA-8′-hydroxylase, encoded by the *CYP707A* gene (a member of the cytochrome P450 family) [101], and ABA can be reversibly inactivated by glucosylation. ABA-glucosylester (ABA-GE) is a physiologically inactive storage and transfer form, and ABA is conjugated with glucose by ABA glycosyltransferase (GT), forming ABA-GE [102], while *β*-glucosidase (*β*-Glu) can convert ABA-GE to ABA [103,104]. In the signaling pathway of ABA, ABI3 (ABA insensitive 3) is a central regulator, and it can interact with and can be polyubiquitinated by AIP2 in vivo; and there are three major components, including the ABA receptor protein (PYR/PYL/RCAR), ABA signaling pathway negative regulator, including ABI1/PP2C (a type 2C protein phosphatase [105,106] and ABI5/DPBF1, which belongs to thew AREB/ABF/bZIP (ABF) subfamily genes and is a transcription activator of ABRE-dependent ABA signaling [107–111].

To better elucidate the molecular mechanism of ABA regulation of fruit cracking, many researchers in related fields began to screen and study the genes related to the ABA metabolism pathway that may be involved in the regulation of fruit cracking. For example, high-throughput comparative transcriptomic analyses of uncracked and cracked pericarp of litchi were carried out [1]. Among other DEGs, there are 21 genes involved in ABA metabolism in the pericarp of cracked and uncracked litchi fruits, including one *LcABI5*, one *LcABI1*, two *LcCYP707A*, two *LcPP2C*, six *Lcβ-Glu* and nine *LcGT* (Table 4). And among them, two *LcCYP707A* genes were upregulated two-fold and five-fold in uncracked litchi compared to cracked fruits, respectively. It was observed that an increase in the expression level of *CYP707A2* resulted in a decrease in ABA content, and the ABA level was six-fold higher in *cyp707a2* mutants than wild-type plants [102]. Two *LcCYP707A* genes were downregulated in cracked litchi fruits, suggesting that ABA may accumulate in cracked litchi fruit. Physiologically active ABA is conjugated with glucose by GT and then forms the inactive ABA-GE; *AtBG1*(a *β-Glu*) contributes to increase the ABA level, and the loss of *AtBG1* leads to a decreased ABA level; and PP2C acts as a negative regulator of ABA signaling [102]. Li et al. (2014) found that nine *LcGT* genes were upregulated between 2-fold and 17-fold, and six *Lcβ-Glu* genes were downregulated between 2-fold and 12-fold in uncracked fruits compared to the cracked fruits of litchi, implying that the ABA content is lower in uncracked litchi fruit. Two *LcPP2C* and one *LcABI1* genes were upregulated 2-fold to 9-fold, and one *LcABI5* gene was downregulated at least 2-fold in uncracked fruits compared to cracked litchi fruits (Table 4), which indicated that the cracking of litchi is related to the genes involved in ABA signaling [1]. In addition, based on the expression levels

of *LcCYP707A* genes and *LcPP2C* genes in the three groups of samples, it was speculated that the ABA levels were higher in the cracked CS variety 'Baitangying' sample compared with the CR variety 'Feizixiao' pericarps [43]. In summary, the upregulated expression of *Lcβ-Glu* and *LcABI5* genes in the pericarp and the downregulated expression of *LcCYP707A*, *LcGT*, *LcPP2C* and *LcABI1* genes in the pericarp could then cause an increased ABA level in the pericarp and eventually to induce fruit cracking in litchi.

**Table 4.** Potentially cracking-related genes involved in the ABA pathway.

| Genes Annotation | Gene Name and Accession | Species | References |
|---|---|---|---|
| Zeaxanthin epoxidase | *LcZEP/Lc.0.938*<br>*PaABA1/Pav_sc0000071.1_g630*<br>*ZjZEP/gene18925* | Litchi<br>Cherry<br>Jujube | [88]<br>[110]<br>[45] |
| 9-cis-epoxycarotenoid dioxygenase | *ZjNCED/gene30271/gene1854* | Jujube | [45] |
| zeaxanthin epoxidase | *ZjZEP/gene18925* | | |
| ABA 8′-hydroxylase | *PaABAH1/Pav_sc0001440.1_g080*<br>*LcCYP707A/Unigene0007266/Unigene0026783*<br>*LcCYP707A/c42183_g1_i1* | Cherry<br>Litchi<br>Litchi | [110]<br>[1]<br>[44] |
| β-glucosidase | *Lcβ-Glu/Unigene0016580/Unigene0016134*<br>*Unigene0018025/Unigene0043976*<br>*Unigene0012400/Unigene0016425*<br>*Lcβ-Glu/Lc.0.11/Lc.0.3431/Lc.0.157/Lc.0.3975* | Litchi<br><br>Litchi | [1]<br><br>[44] |
| Glycosyltransferase | *LcGT/Unigene0042108/Unigene0002939*<br>*Unigene0038887/Unigene0001499/Unigene0011269/*<br>*Unigene0028438/Unigene0042417/*<br>*Unigene0002586/Unigene0027109* | Litchi | [1] |
| Glycosyltransferase | *LcGT/Lc.12.1389* | Litchi | [44] |
| ABA insensitive | *LcABI1/Unigene0027077*<br>*PaABI1.1/Pav_sc0000069.1_g410*<br>*PaABI1.2/Pav_sc0000129.1_g370*<br>*PaABI1.3/Pav_sc0000212.1_g830*<br>*PaABI1.4/Pav_sc0000689.1_g430*<br>*PaABI1.5/Pav_sc0000689.1_g440*<br>*LcABI5/Unigene0037679*<br>*PaABI5/Pav_sc0000363.1_g920* | Litchi<br>Cherry<br><br><br><br><br>Litchi<br>Cherry | [1]<br>[110]<br><br><br><br><br>[1]<br>[111] |
| ABA receptor protein | *PaPYL1/Pav_sc0001428.1_g450*<br>*PaPYL4/Pav_sc0001341.1_g250*<br>*PaPYL8/Pav_sc0001335.1_g500*<br>*PaPYL9/Pav_sc0000591.1_g120*<br>*PaPYL12/Pav_sc0000037.1_g470* | | |
| Protein phosphatase 2C | *ZjPP2C/gene7093*<br>*LcPP2C/Unigene0009174/Unigene0047715* | Jujube<br>Litchi | [45]<br>[1] |
| Protein C2-domain ABA-related 4/7 | *PaCALB 4/Pav_sc0000103.1_g680*<br>*PaCALB 7/Pav_sc0000221.1_g240* | Cherry | [110] |
| ABRE binding factor | *PaABF2/Pav_sc0000852.1_g810*<br>*PaABF3/Pav_sc0002234.1_g130* | | |
| ABRE binding protein 3 | *PaAREB3/Pav_sc0001836.1_g030* | | |
| ABA overly-sensitive 5 | *PaABO5/Pav_sc0000015.1_g160* | | |
| ABA deficient 4 | *PaABA4/Pav_sc0000409.1_g020* | | |
| ABA binding protein | *PaFCA/Pav_sc0000028.1_g190* | | |
| ABA-aldehyde oxidase isoform | *PaAAO3/Pav_sc0001251.1_g340* | | |
| ABA-responsive family protein | *PaHVA22/Pav_sc0002080.1_g050* | | |
| Respiratory burst oxidase homolog protein D | *ZjRBOHPD/gene16443* | Jujube | [45] |
| Serine/threonine-protein kinase | *ZjSAPK1/gene17084* | | |
| Glycogen synthase kinase | *LcGSK/Lc.0.1659* | Litchi | [88] |
| NAC domain protein | *MdNAC058/MDP0000246482* | Apple | [68] |

In recent years, it was found that changes in gene expression levels and metabolites can explain the cracking susceptibility of fruits (Table 4); for example, an upregulation in the *LcCYP707A* (*c42183_g1_i1*) gene involved in ABA metabolism was found in the CR litchi cultivar 'Feizixiao' [43]. In addition, it was observed that one *LcZEP* (*Lc.0.938*) gene and genes encoding one GT family protein (*LcGT*, *Lc.12.1389*), four *β*-Glu (*Lc.0.11*, *Lc.0.3431*,

*Lc.0.157* and *Lc.0.3975*) and one GSK (*Lc.0.1659*) involved in ABA signaling were more highly expressed in the CS cultivar 'Nuomici' than in the CR cultivar 'Huaizhi' aril, implying that the increased ABA content and these osmotic regulation substances might be higher in the 'Nuomici' aril than in the CR cultivar 'Huaizhi' aril [88]. In the study of jujube, the cracked and uncracked fruits of the CS cultivars 'Cuizaohong' and 'Jinsixiaozao' and uncracked fruits of the CR cultivar 'Muzao' were selected for comparative transcriptome analyses, and the expression levels of two ABA related genes (Table 4), one *ZjNCED1* (*gene30271*) and one Zj*RBOHPD* (*gene16443*) gene were upregulated in the cracked samples of 'Cuizaohong' and 'Jinsixiaozao' compared to the cracked samples, respectively. And five ABA metabolism genes, including two *ZjNCED1* (*gene30271* and *gene1854*), one *ZjZEP* (*gene18925*), one *ZjPP2C* (*gene7093*) and one *ZjSAPK1* (*gene17084*) (Table 4) were gradually downregulated in the cracked compared with uncracked fruit samples of 'Jinsixiaozao' and non-cracking 'Muzao' [45]. In addition, it was found that *NAC058* (Table 4), a gene involved in ABA signaling, is upregulated during the development of apple fruits and may prevent cracking initiation [68]. In sweet cherry, the transcriptional level of genes related to ABA metabolism or signaling, such as *PaPYL1/4/8/9/12*, *PaABA1*, *PaABI1.2*, *PaAREB3*, *PaHVA22* (Table 4), were downregulated in the skin and flesh of the CR cultivar 'Regina' compared to the CS cultivar 'Early Bigi' [110], while *PaAAO3*, *PaABAH1*, *PaABI1.1/1.3/1.4/1.5/5*, *PaABF2/3*, *PaABO5*, *PaFCA*, *PaCALB4/7* and *PaABA4* (Table 4) were upregulated in the skin and flesh of the CR variety 'Regina' compared to the CS variety 'Early Bigi' [110]. Thus, the above genes may be significant ABA metabolism and signaling pathway genes involved in mediating fruit cracking or splitting, and the roles of genes regulated or related to ABA need to be further studied and considered while developing cracking-resistant varieties through fruit breeding programs.

## 5. Discussion

Several fruits species are highly influenced by fruit cracking or splitting, a common and severe physiological disorder that severely compromises the fruit appearance and diminishes fruit quality, shelf life and market value, enhances microbial infections and causes immense economic losses to fruit producers. Cracking or splitting cannot be attributed to a single influencing factor, and it is difficult to study cracking in vitro under controlled conditions due to lack of the experimental methods to induce fruit cracking. Although fruit cracking has been investigated for nearly 100 years, and there have been many research studies carried out to investigate the possible reasons and control methods of cracking in many fruit crops, the detailed physiology and molecule mechanisms of cracking are still poorly understood, and it remains a significant challenge for fruit producers and researchers, becoming a research hotspot globally, attracting more and more people to engage in the study of fruit cracking.

It is well known that fruit cracking is the result of the split of the fruit peel surface and the outer flesh around the calyx during rapid cell enlargement [26], which is a very complex physiological disorder process and is closely associated with the homeostasis of endogenous plant hormones. In recent years, advancements in several fleshy fruits' genome research have promoted the emergence and development of functional genomics and provided significant opportunities and challenges to explore, analyze and clarify the underlying genetic mechanisms governing fruit cracking. And with the help of multi-omics-based biological research technologies, including genetics, epigenetics, transcriptomics, proteomics, metabolomics, molecular biology, physiological and biochemical methods, we have witnessed remarkable progress on fruit cracking at the molecular level. And the genetic component has been discovered and makes fruit cracking an attractive field for researchers who work with the molecular breeding of fruit crops, and breeders have incorporated the molecules into practical breeding strategies, including gene editing, transgenic approaches and progressive breeding, which holds the potential for achieving molecular-design breeding and the efficient genetic enhancement of fruit crops, and developed crack-resistant cultivars in many fruit species, while only minor advances have

been made in understanding the physiology and molecular mechanisms of fruit cracking in any of these fruit crops. In turn, this has made it difficult to recommend reasonable and effective preventive measures for fruit cracking.

ABA is a stress-responsive hormone that inhibits seed germination and seedling growth to adapt to an unfavorable environment, while GA is a major growth-promoting hormone that promotes seed germination, seedling growth, flowering and leaf expansion [110–112]. It is well known that GA and ABA have antagonistic functions on many aspects of regulating plant growth and development processes and responses to biotic or abiotic stresses in higher plants, especially seed dormancy or germination, which mainly depends on the balance of the two hormone signals of GAs and ABA. Previous studies have reported the main components of the ABA and GA signaling pathways and shown that DELLA proteins represent a vital regulatory hub that mediates the repression of ABA on GA signaling in higher plants [113–116]. In addition, studies have observed that ABA can antagonize the GA-promoted degradation of DELLA proteins [117], and the results presented in the study of Lin et al. (2015) suggested that the SnRK2s-APC/CTE regulatory module represents a new and significant signaling hub regulating the antagonistic function of GA on ABA signaling in plant crops [63]. Favorable environmental conditions can activate the GA pathway, promoting the degradation of ABA core signaling components (ABA receptors and SnRK2s) and GA signal suppressors (DELLA proteins) [63] and promoting plants to resume growth and development healthily. The antagonistic action of GA and ABA thus may serve as a 'rheostat' to fine tune the growth and development of plants in response to fluctuating environmental conditions. Considering these facts, it is notable that the employment of multiple E3 ligases for the proteasomal degradation of the DELLA proteins and ABA signaling components possibly allows the plants to more effectively respond to different developmental or external signals. Thus, identification and functional studies of other unknown E3 ligases will lead to a better understanding of GAS and ABA signaling mechanisms and the crosstalk with other signaling pathways in regulating fruit cracking.

## 6. Conclusions and Perspectives

### *6.1. Conclusions*

It has been preliminarily demonstrated that the growth-promoting plant hormone GAs and growth-inhibiting plant hormone ABA play important regulatory roles in governing fruit cracking. And fruit cracking is directly correlated with the higher GA and ABA levels in fruits [117,118]. During the process of fruit cracking, the expression levels of many genes are precisely regulated by the endogenous phytohormones GAs and ABA to cope with the transition of fruit development and cracking state and the change in environmental conditions [1,45,63,68,119]. And recently, publications have suggested GAs- and ABA-related and -regulated gene-specific expression plays a crucial role in cracking development, namely GA-pathway-related genes, including *KS*, *GA2ox*, *GA20ox*, *GID1*, *GPR*, and ABA pathway related genes, including *ABI*, *ABF*, *ABO5*, *FCA*, *ABI1*, *ABI5*, *b-Glu*, *GT*, *CYP707A*, *PP2C*, *ZEP*, *NAC058* and so on [1,43,44,68,86,109]; these may be the major candidate genes that regulate fruit cracking, and they can be further applied in molecular breeding efforts to produce improved crack-resistance that meet the demands of modern fruit crops' production.

### *6.2. Perspectives*

With the completion of several fruit genome sequencing studies and the identification of more GAs and ABA mutants, there are more and more studies on the key genes of ABA and GAs regulating fruit cracking. Even so, the molecular cues related to cracking are mainly based on correlations due to the direct proof of cracking based on mutations or reverse genetics still being missing and the regulatory mechanisms underlying the antagonism of GAs and ABA signaling pathways in fruit cracking control remaining largely unknown. It is still unclear whether GAs and ABA have antagonistic effects on

the regulation of fruit cracking. And how do phytohormone GAs and ABA control fruit cracking through regulatory networks? These details need further genetic analysis and molecular identification to clarify. Yeast one-hybrid (Y1H) and yeast two-hybrid (Y2H) techniques were used to isolate and identify the transcription factors that combine the GA response element (GARE) and ABA response element (ABRE) in the signaling pathway of the ABA- and GA-regulated growth and development of plant crops and then to study their functions and interactions; this approach can promote the study progress of the fruit cracking mechanism and provide new ideas and research methods for fruit cracking resistance breeding.

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