Advances in Molecular Mechanisms of Plant Flower and Fruit Abscission: Postprint

Authors: CUI Yasong; CHEN Qingfu; HUO Dongao; LI Hongyou

Date: 2018-06-22T00:00:00+00:00

Abstract

Flower and fruit abscission is a ubiquitous natural phenomenon involving the detachment of flowers, fruits, and seeds from the maternal plant. The region where organ abscission occurs is designated as the abscission zone (AZ). The abscission zone differentiates to form the abscission layer, which is intimately associated with the abscission process. The development and functional execution of the abscission layer constitute a complex and precise process involving multiple enzymes, hormones, and genes. Flower and fruit abscission represents not only a typical agronomic trait in crop cultivation and breeding but also constitutes one of the primary forms of plant organ abscission. Reducing flower and fruit abscission in plants or controlling moderate abscission in certain species to improve the yield and quality of crops and horticultural plants represents a key objective in crop domestication efforts. This article, based on previous research into the physiological, biochemical, and molecular biological mechanisms of plant organ abscission, primarily elucidates the molecular mechanisms of flower and fruit abscission from the perspectives of cytological basis, physiological and biochemical mechanisms, genetic principles, molecular biology, related gene mapping, and transcriptome analysis, with particular emphasis on dissecting the functional mechanisms of abscission from the molecular biological and gene mapping aspects, thereby providing theoretical guidance for crop genetic breeding research.

Full Text

Preamble

DOI:10.11931/guihaia.gxzw201710013

Research Progress on the Molecular Mechanism of Plant Flower and Fruit Abscission

CUI Ya-Song, CHEN Qing-Fu*, HUO Dong-Ao, LI Hong-You
(Research Center of Buckwheat Industry Technology, Guizhou Normal University, Guiyang 550001, Guizhou, China)

Abstract

Flower and fruit abscission is a widespread natural phenomenon involving the detachment of flowers, fruits, and seeds from the mother plant. The region where organ detachment occurs is called the abscission zone (AZ). The abscission layer differentiates within the AZ and is closely related to the shedding process. The development and functional execution of the abscission layer constitute a complex and precise process involving multiple enzymes, hormones, and genes. Flower and fruit abscission represents not only a typical agronomic trait in crop cultivation and breeding but also one of the primary forms of plant organ shedding. Reducing excessive abscission or controlling moderate shedding in certain plants to improve yield and quality has been a long-standing goal in crop domestication. Based on previous studies on the physiological, biochemical, and molecular mechanisms of plant organ abscission, this review elucidates the molecular mechanisms of flower and fruit abscission from perspectives including cytological foundations, physiological and biochemical mechanisms, genetic patterns, molecular biology, gene mapping, and transcriptome analysis, with particular emphasis on molecular biology and gene mapping to provide theoretical guidance for crop genetics and breeding research.

Keywords: abscission layer, flower and fruit abscission, cytology, gene mapping, molecular mechanism

Introduction

Flower and fruit abscission is a ubiquitous natural phenomenon where flowers, fruits, and seeds detach from the main plant body. During normal growth and development, plants may undergo normal or abnormal organ shedding to adapt to environmental stresses (such as drought, flooding, extreme temperatures, nutrient deficiencies of N, B, Ca, Zn, pests, and diseases) or to balance their growth by reducing burden or dispersing seeds (propagules). Normal shedding, such as leaf abscission, helps conserve water and reduce pathogen infection (Patharkar et al, 2015; Faeth et al, 1981), while normal flower and fruit abscission regulates source-sink relationships and promotes rational distribution of nutrients and minerals. However, abnormal shedding inevitably causes significant losses in nature, such as seed shattering in rice and wheat, boll shedding in cotton, pod abscission in soybean, and flower/fruit drop in fruit trees, all leading to substantial yield reductions. In agricultural production, rational regulation of organ abscission can effectively utilize land resources and help crops achieve high yield and quality.

Sun et al (2016) reported that foliar application of the plant growth regulator DTA-6 reduced the expression of GmAC in soybean pod abscission zones,

altered physiological metabolism in these zones, and decreased pod abscission rates. Anthony & Cogginsjr (1999) and Yuan & Carbaugh (2007) applied indole-3-acetic acid (IAA) and ethylene (ETH) inhibitors before harvesting citrus and apple to prevent pre-harvest fruit drop. Researchers have identified key loci controlling shattering traits in staple crops such as soybean and sorghum through genetic analysis of domestication-related traits using molecular markers (Lin et al, 2012). In tomato, the jointless phenotype facilitates post-harvest handling and reduces damage from residual pedicels, thereby improving fruit quality (Mao et al, 2000). Molecular marker-assisted breeding and gene pyramiding are effective approaches for improving breeding efficiency, and reviewing the physiological and molecular mechanisms of abscission zone formation can provide valuable insights for both mechanistic research and breeding practice.

1. Cytological Basis of Flower and Fruit Abscission

Bleecker et al (1997) proposed that plant flower and fruit abscission is closely associated with abscission layer formation, and Patterson et al (2004) subsequently discovered that rice seed abscission is related to the abscission layer between the spikelet and pedicel. The process is generally considered to involve four stages (Figure 1 [Figure 1: see original paper]): First, abscission zone cells are formed; second, the abscission zone receives abscission signals and initiates shedding; third, the zone perceives these signals, undergoes enzymatic hydrolysis, and cell separation occurs; and finally, abscission zone cells differentiate to form the abscission layer, while a protective layer forms adjacent to the abscission layer cells (on the plant body side) after organ detachment (Kim, 2014; Wang et al, 2017). Notably, some plant organs shed without forming a protective layer, such as soybean compound leaves (Moline & Bostrack, 1972).

Research on the first stage of abscission layer formation is relatively extensive. Nakano et al (2012) demonstrated that plants differentiate an abscission zone (AZ) at the base of organs destined to abscise. The AZ comprises the tissue region and adjacent areas where detachment occurs, typically consisting of 5-50 layers of small, isodiametric cells with dense cytoplasm and small intercellular spaces. Despite the presence of numerous AZ cells, only 1-2 cell layers actually separate during the abscission process, forming the separation layer. Sexton et al (1982) explained this phenomenon by suggesting that inductive factors are only effective at the separation layer, and while all abscission layer cells have the potential to differentiate, only 1-2 layers are sufficiently sensitive to the induction signals. The number of abscission layer cell layers varies among different plants and tissues: Arabidopsis thaliana floral organs have 4-6 layers (McManus et al, 1990; Patterson et al, 2004), rice has 12 layers (Zheng, 2016), and elderberry (Sambucus nigra) leaf abscission zones have 30 layers (Taylor, 2001).

Doorn & Stead (1997) categorized abscission zone cells into three types based on morphological comparisons: cells with similar morphology and isodiametric size; cells smaller than neighboring cells and rectangular in shape; and cells similar

in morphology and size to adjacent cells. Bair (1984) observed in Lamiaceae (Coleus) under electron microscopy that abscission zone cells had smaller vacuoles, larger nuclei with prominent nucleoli, increased organelles (polyribosomes, Golgi bodies, rough endoplasmic reticulum, and mitochondria), and invaginated cell membranes with paramural bodies. Recently, Tang & Chen (2017) conducted comprehensive analyses of pedicel articulation in seven shattering and non-shattering buckwheat species using paraffin sectioning and anatomical observation. They found that during buckwheat flowering, parenchyma cells in the pedicel contained abundant starch granules, vascular bundles were poorly differentiated, and cells were small and tightly arranged—features similar to previously studied abscission layer structures. This suggests that pedicel articulation functions similarly to abscission layers, and when the pedicel shows constriction, fruit abscission typically occurs.

2. Physiological Basis of Flower and Fruit Abscission

2.1 Physiological Basis of IAA and ETH Regulation

IAA and ETH play crucial roles in plant flower and fruit abscission (Eo & Lee, 2009; Meir et al, 2010; Patterson & Bleecker, 2004; Iglesias et al, 2004). The IAA signaling pathway in the abscission zone is key to inhibiting organ shedding. Both Addicott et al's (1955) "auxin gradient theory"and subsequent applications of exogenous plant growth regulators (-naphthaleneacetic acid, NAA; IAA; 2,4-dichloroacetic acid, 2,4-D) to different plants or different parts of the same organ have demonstrated abscission inhibition in crops such as tomato, soybean, citrus, apple, and pear, as well as in tomato petiole distal and proximal ends. These experiments laid the foundation for studying flower and fruit abscission.

Generally, the auxin concentration at both ends of the abscission zone directly affects organ shedding, with polar auxin transport to the zone being the primary condition for blocking abscission layer formation (Taylor & Whitelaw, 2001). IAA negatively regulates organ abscission by modulating abscission zone cell insensitivity to ETH (Sexton & Roberts, 1982). Roberts et al (2000) showed that IAA delays abscission layer formation while ETH promotes it. Brown (1997) suggested that IAA inhibits organ abscission because when leaves experience environmental stress, IAA flow to the abscission zone decreases, making the zone more sensitive to ETH (Sakamoto et al, 2008). Consequently, applying the ethylene inhibitor aminoethoxyvinyl glycine HCl combined with synthetic auxin 2,4-dichlorophenoxyacetic acid can prevent pre-harvest fruit drop in citrus and apple (Anthony & Coggins, 1999; Yuan & Carbaugh, 2007).

Jasmonic acid (JA) also inhibits Arabidopsis floral organ abscission. Ueda et al (1996) found in common bean that JA increases cellulase activity, hydrolyzing polysaccharides in cell walls at the leaf petiole base and promoting abscission. Kim et al (2013) discovered that mutation of the JA receptor coronatine insensitive1 (coi1) delayed Arabidopsis floral organ abscission. In addition to IAA and JA, other hormones such as ABA and salicylic acid show promise in regulating

senescence, as both floral organs and leaves appear to senesce before abscission (Guiboileau et al, 2010; Patharkar & Walker, 2015, 2016). ETH and GAs, which promote abscission, have been identified in Arabidopsis cell separation layers (Arnaud et al, 2010).

2.2 Physiological Basis of Key Enzyme Regulation

During physiological processes such as soybean pod dehiscence, Arabidopsis petal abscission, tomato flower pedicel shedding, and shattering in buckwheat, rice, and wheat, numerous intriguing but poorly understood events occur in the AZ once abscission is activated. As abscission progresses, AZ cells enlarge and the cytoplasm becomes more alkaline, as demonstrated in Arabidopsis and tomato (Sundaresan et al, 2015), though the cause of this pH change remains unclear. One hypothesis suggests that alkalinity may represent the optimal pH for abscission enzymes.

Many enzymes increase markedly within abscission zone cells, including amino acids, proteins, mRNA, and rRNA. RNAs in the AZ are ultimately translated into proteins or enzymes, among which cellulases and pectinases are the most important. Increased cellulase activity in the abscission layer persists until final detachment, with cellulase isozymes and pectinases working together during growth and development to facilitate shedding. -1,4-glucanase activity in the abscission zone shows significant increases and upregulated expression during abscission. Another important change is the degradation of pectin calcium salts and other pectin substances in the middle lamella into pectin and pectic acid. Notably, some enzymes (polygalacturonase, glucanase) do not participate in AZ formation but instead degrade pectin and cellulose in cell walls to promote abscission. Other enzymes such as RNases, DNases, proteases, phosphatases, phenylalanine ammonia-lyases, and peroxidases may promote abscission by facilitating protective layer formation in the abscission zone and accelerating wound lignification. Based on this, spraying phenylalanine ammonia-lyase inhibitor AIP (2-aminoindan-2-phosphonic acid) on fruit pedicels reduces phenylalanine ammonia-lyase activity and decreases fruit abscission rates.

During abscission, certain pathogenesis-related proteins also increase significantly, including chitinases, expansins, pathogenesis-related proteins, metallothioneins, and WRKY proteins expressed in the abscission zone (Cho et al, 2000; Belfield et al, 2005; Meir et al, 2010; Li, 2014). Notably, the LX protein, a key protein in programmed cell death (PCD) and a T2/S-like ribonuclease, shows specific expression in mature tomato abscission zones with asymmetric expression in proximal and distal ends, representing a critical physiological and biochemical change for pedicel abscission (Bar et al, 2011). In Arabidopsis, the PCD-related nuclease BFN1 is also induced in abscission zones. Interestingly, the Arabidopsis BFN1 promoter is expressed in other tomato tissues including leaves, flowers, and fruits (Lers et al, 1998; Lehmann et al, 2001; Lers et al, 2006).

3. Genetic Patterns of Flower and Fruit Abscission

3.1 Traditional Genetic Patterns

From a traditional genetics perspective, spontaneous or artificially induced mutations in plant genomes alter abscission phenotypes, and these phenotypic changes can be traced to corresponding mutant genes. Traditional genetics has primarily focused on flower abscission mechanisms, such as tomato abscission zone formation genes (jointless, J2, LS). Mutation of the J gene in tomato causes deletion of 939 bp including part of the first exon and the upstream region of the start codon, transforming tomato from a variety with abscission zones to one without. The jointless mutant phenotype shows non-abscising pedicels (Mao et al, 2000; Roldan et al, 2017). In tomato 1,4- -glucanase (cel2) antisense transgenic plants, the breaking strength of fruit abscission zones significantly increases (Brummell et al, 1999). Yang et al (2005) further demonstrated that the J2 gene near the centromere of tomato chromosome 12 also controls abscission zone formation. Schumacher et al (1999) found that the absence of petals in tomato LS mutants was primarily caused by mutation of the LS gene regulating abscission zone development. The Arabidopsis gene AtLAS, homologous to tomato LS, shows delayed floral organ abscission in its knockout mutant las (Greb et al, 2003). In Arabidopsis double mutants bop1 and bop2, failure to form abscission zones results in absent floral organ abscission phenotypes, indicating that Bop1 and Bop2 genes are involved in abscission layer development (McKim et al, 2008). Wang et al (2006) demonstrated through gene knockout experiments that KNAT/BP regulates Arabidopsis floral organ abscission zone development, with bp mutants showing increased bubble cells in floral organ abscission zones and premature organ shedding.

Not all delayed abscission phenotypes result from abscission zone developmental regulation. Jinn et al (2000) found that antisense suppression of the Arabidopsis receptor kinase HAESA (a leucine-rich repeat receptor) delayed petal abscission in outer whorls, with severe transgenic lines showing no petal abscission. Stenvik et al (2008) proved that IDA-derived peptides regulate Arabidopsis floral organ abscission through HAESA. Additionally, IDA receptor genes (HAESA, HAE) are not affected by exogenous ethylene, and knockout mutants of HAE and its homolog HAESA-LIKE 2 (HSL2) show significantly delayed floral organ abscission (Jinn et al, 2000; Patharkar & Walker, 2016). The Arabidopsis auxin response factor 2 (ARF2) mutant obtained through T-DNA insertion technology may delay abscission zone cell separation by inhibiting cell wall-degrading enzyme activities, thereby delaying floral organ abscission (particularly stamens) but not preventing it. Interestingly, arf1 can enhance the abscission phenotype of arf2 mutants, indicating that arf1 and arf2 have partially redundant functions in regulating abscission. Other Arabidopsis mutants also show interactions, as mutations in ARF1, NPH4/ARF7, and ARF19 can enhance the delayed abscission phenotype of arf2 mutants (Ellis et al, 2005; Okushima et al, 2005). Guan (2015) found delayed floral organ abscission in SLARF2-RNAi plants. Ethylene receptor mutants are also noteworthy, as ethylene receptor

mutants etr1 and downstream mutants ein2, ein3, and ers2 obtained through EMS mutagenesis show delayed floral organ abscission (Patterson & Bleecker, 2004). When ethylene receptor ETR1 binds external ethylene signals, it activates downstream EIN2, which subsequently activates transcription factor EIN3 to regulate downstream target genes and transmit ethylene signals, accelerating plant organ abscission.

Additionally, the Arabidopsis ACTIN-RELATED PROTEINS (ARP) family plays a critical role in floral organ abscission. Kandasamy (2005) observed significantly delayed floral organ abscission in ARP7 knockout transgenic lines generated through RNAi experiments, while abscission zone development and ethylene triple response remained consistent with wild type, indicating that ARP7 regulates floral organ abscission independently of the ethylene pathway. Similar abscission-regulating genes include AGL15, HAESA, and ARP4. Cho & Cosgrove (2008) discovered that sequential action of IDA, HAESA, HSL2 (HAESA-LIKE2), and the MAPK cascade signaling pathway controls Arabidopsis floral organ abscission. For example, floral organs cannot abscise when HAESA downstream components including MKK4/5 mitogen-activated protein kinase (MAPK) cascade and MPK3/6 MKK cascade are disrupted, whereas activated MKK4/5 expression can restore the abscission phenotype of hae/hsl2 double mutants, demonstrating that MKK4/MKK5 activation of MPK6/MPK3 controls Arabidopsis floral organ abscission. It remains unclear which MAP triple kinase functions in the abscission pathway and whether other intermediates exist between the HAE receptor complex and MAPK cascade.

Patharkar & Walker (2015) surprisingly found that MKK4/5 knockout reduces normal HAE expression to less than 20%, as HAE is considered an upstream gene of the MAPK cascade when abscission is activated in floral organ abscission zones. Overexpression of the MADS domain transcription factor AGAMOUS-LIKE 15 (AGL15) blocks abscission without altering abscission zone development, indicating that AGL15 is a negative regulator of abscission (Fernandez et al, 2000) (Figure 2 [Figure 2: see original paper] and Table 1). Once the abscission signaling pathway is activated, the MAPK cascade phosphorylates AGL15 at serine residues 231 and 257, inhibiting HAE expression. Once newly synthesized HAE replaces the plasma membrane, a positive feedback loop is completed. Both the positive feedback network and MAPK cascade significantly amplify the initial discrete signals, explaining the increased HAE expression during floral organ abscission. Although AGL15 appears to be the primary transcription factor regulating abscission, it is not the only one. For instance, agl15/agl18 double mutants abscise earlier than wild type, indicating that AGL15' s sister protein AGL18 plays a partially redundant role (Patharkar & Walker, 2015; Patharkar et al, 2016).

Normal abscission requires ADP-ribosylation factor GTPase-activating protein (NEV, NEVERSHED). NEV mutations alter Golgi apparatus structure and trans-Golgi network positioning. nev mutants accumulate numerous vesicles in paramural regions, likely resulting from trafficking of HAE and other proteins

to the plasma membrane (Liljegren et al, 2009). Mutations in three different suppressor genes can partially restore vesicle trafficking and abscission in nev mutants. The first suppressor gene is EVERSHED (EVR), also known as SUPPRESSOR OF BIR1 (SOBIR1, BIR11), a receptor-like protein kinase (Leslie et al, 2010). Mutations in BAK1-INTERACTING RECEPTOR-LIKE KINASE 1 (BIR1) can suppress pathogen responses through secondary mutations in EVR/SOBIR1 (Gao et al, 2009), and secondary mutations in SERK1 can also suppress the nev phenotype (Lewis et al, 2010). From a molecular mechanism perspective, how mutation of HAE co-receptors can restore abscission in nev mutants remains unclear. The triple mutant serk1/serk2/bak1 actually shows a mild floral organ abscission deficiency phenotype (Meng et al, 2016) (Table 1). Finally, secondary mutations in CAST AWAY (CST), a receptor-like cytoplasmic kinase, can also suppress the nev mutant phenotype. CST interacts with HAE and EVR in Arabidopsis leaf mesophyll protoplasts (Burr et al, 2011). Secondary mutations in EVR, SERK1, and CST can all restore abscission in nev mutants, but the final abscission zone scars in these plants are over-differentiated, suggesting that some genes involved in abscission activation also function in final scar differentiation. In addition to vesicle shuttling, HAE can detect endoplasmic reticulum (ER) errors, and the ER-associated degradation (ERAD) system ensures HAE quality control (Baer et al, 2016). When the ERAD system is defective, HAE alleles producing partially functional proteins can still reach the plasma membrane and transduce abscission signals. Figure 2 presents a model of the abscission activation signaling pathway.

3.2 Modern Genetic Patterns

Modern genetics (molecular biology at the gene level) differs from traditional genetics in abscission research by focusing primarily on fruit abscission patterns, which are more complex and not simply controlled by single or few genes as qualitative traits. Porter (1959) inferred from Cimarron×Wichita wheat crosses that shattering is controlled by recessive genes among multiple loci. In Blackhull×Wichita populations, shattering is controlled by two gene pairs, with spikelet abscission primarily related to the abscission layer, whose development is controlled by the Q locus on the rachis, and the number of abscission cell layers depends on expression of the recessive allele q at this locus (Simons et al, 2006; Zhang et al, 2011; Sormacheva et al, 2015). Additionally, common buckwheat (Fagopyrum esculentum) shattering is controlled by at least two gene pairs, later found to be regulated by three dominant genes, with shattering occurring when any two dominant genes are complementary (Wang et al, 2005). Studies revealed that the homostylous self-fertile gene H is tightly linked with one shattering gene Sht in sweet buckwheat (Yasui et al, 1987; Pan & Chen, 2010). In breeding practice, crosses between non-shattering self-fertile buckwheat lines can produce shattering hybrid offspring, suggesting that at least two dominant complementary genes control shattering inheritance. Yue et al (2012) found that shattering follows a 9:7 segregation ratio in two F2 populations from reciprocal crosses between sweet buckwheat lines 21-1 and Lorena-3, indicating

a two-gene dominant complementary inheritance pattern. When both parents show intermediate shattering, their hybrid offspring often segregate according to quantitative inheritance patterns controlled by multiple genes. Shattering control involves both major and minor genes, with different shattering intensities observed among types, clearly indicating quantitative trait inheritance.

4. Molecular Biology of Flower and Fruit Abscission

4.1 Molecular Mechanisms of IAA and ETH Regulation

IAA primarily affects the abscission process by regulating gene expression through ARF proteins, which generally consist of a DNA-binding domain (DBD), middle region (MR), and C-terminal domain (CTD) (Hagen & Guilfoyle, 2002). The middle region contains repressors (proline, serine, and threonine) and activators (glutamic acid, leucine), enabling activation or inhibition of auxin-regulated gene transcription. The DBD specifically binds to the TGTCTC sequence of AuxRE (auxin response element) elements to regulate IAA physiological effects (Ulmasov et al, 1999a; Wu et al, 2011). Gray & Estelle (2000) found that these effects vary with IAA concentration: at low IAA levels, the DBD region at the N and C termini of ARF proteins can bind with Aux/IAA to form Aux/IAA-ARF complexes that inhibit ARF activity; at high IAA levels, IAA binds TIR1, promoting Aux/IAA ubiquitination and degradation, thereby activating ARFs and inducing auxin-responsive gene expression.

Meir et al (2010) analyzed the transcriptome of tomato flower abscission zones in response to auxin and found that expression of some Aux/IAA family genes decreased during flower abscission. Arabidopsis auxin response factor genes ARF1, ARF2, ARF7, and ARF19 are all involved in floral organ abscission zone formation. These studies primarily demonstrate that ARF genes regulate abscission zones through inhibition, with auxin suppressing abscission and ARFs responding to auxin to regulate organ abscission zone formation. Conversely, Gao et al (2016) demonstrated through gene silencing that RhIAA16 gene overexpression in Arabidopsis plays an important role in delaying flower abscission, likely by interacting with ARFs to affect abscission zone development.

In contrast to IAA, ETH not only accelerates senescence and promotes abscission but also induces synthesis of hydrolytic enzymes such as cellulases and pectinases in abscission zones. Parra et al (2011) showed that ETH content directly affects floral organ abscission and accelerates abscission layer formation in various species (Arabidopsis, tomato) and organs (leaves, petals, flowers, fruits). Ethylene induces RNA and protein synthesis in abscission zones, as well as expression of organ abscission-related genes and ethylene-responsive genes (pathogenesis-related proteins, PR). Plant organ abscission-related PR gene expression is also upregulated by ethylene induction (Tucker et al, 2002). Recently, Chen et al (2011) found that overexpression of the MADS transcription factor FOREVER YOUNG FLOWER (FYF) in the ethylene transduction pathway

delays organ abscission.

During abscission zone formation, the zone first perceives abscission signals and then initiates shedding, which explains the antagonistic relationship between ETH and IAA. Two viewpoints exist regarding this antagonism: In the first stage (signal perception), the abscission zone is only sensitive to IAA and insensitive to ETH, while in the second stage (abscission initiation), abscission zone cells respond to ETH but become insensitive to IAA (Sexton et al, 1982; Gao et al, 2013). However, IAA-induced abscission inhibition cannot be reversed by ETH. IAA in the abscission zone may alter plant cell sensitivity to ethylene through an intermediate–ethylene receptors (ETR/ERS1) (Sexton et al, 1982). Roberts et al (2002) and Meir et al (2010) further demonstrated that ethylene receptors ETR/ERS1 may be regulated by IAA to induce timely responses to ethylene signals in abscission zone cells.

4.2 Molecular Mechanisms of Key Enzyme Regulation

 -glucosidase (BG) in cellulases plays a critical role in flower and fruit abscission. As early as 1994, Lashbrook et al isolated two genes (TomCel1, TomCel2) encoding endo-1,4- -glucanases (EGases) from a ripe tomato fruit cDNA library, with their encoded products sharing 50% homology. Additionally, soybean abscission zone-expressed proteins share 68% homology with TomCel1, and avocado (Persea americana Mill.) fruit-expressed AvoCel1 protein shares 57% homology with TomCel2. These two tomato genes show overlapping expression, with both TomCel1 and TomCel2 detectable in the same plant tissues, though expression levels differ by location: abscission zones, mature pollen sacs (TomCel1 mRNA), and ripe fruits (TomCel2 mRNA). Ouyang et al (2007) studied EGase functions in plant cell growth and development, finding that the TomCel2 gene encoding -glucanase is highly expressed in tomato fruit pedicel abscission zones, promoting pectin layer hydrolysis and fruit abscission during cell separation. Restricting TomCel2 expression reduces flower abscission. Campillo & Bennett (1996) isolated six cellulase genes from tomato, among which Cel1, Cel5, and Cel6 showed significant expression during abscission, albeit at different levels, indicating that cellulase regulation of abscission zone formation and organ shedding is a complex process.

Polygalacturonase (PG) and pectin methylesterase (PME) in pectinases also play crucial roles in abscission. Numerous PG genes have been identified as upregulated during plant organ abscission, including tomato TAPG1-TAPG6 (Hadfield et al, 1998), oilseed rape CAW471 (Whitelaw et al, 2002) and RDPG (Zhang, 2013), soybean SDPG (Zhang, 2013), Arabidopsis ADPG1 and ADPG2 (Ogawa et al, 2009), and litchi LcPG1 (Peng et al, 2013). These genes specifically encode endo-polygalacturonases (PGs) expressed in floral organ abscission zones and fruit pedicels. ADPG1 and ADPG2 promote enzymatic hydrolysis and are essential for pod dehiscence, with mutations causing indehiscent fruit formation (Ogawa et al, 2009). Pectin methylesterases localize between plant cell walls and cells (Micheli, 2001). Zhang et al (2012) identified a pectin methylesterase-

encoding gene, At4g022330, expressed in Arabidopsis petals that may participate in pectin metabolism and affect petal abscission. Recently, Kumpf et al (2013) found through lateral root mutant analysis that IDA and HAE regulate PG activity in pectin to control lateral root abscission zone development, with microarray hybridization indicating that IDA-HAE/HSL2 signaling indirectly regulates abscission zone-related genes by modulating cell wall remodeling genes.

4.3 Gene Mapping for Abscission Traits

Molecular mechanisms and gene mapping for seed/fruit shattering and pod dehiscence are crucial for variety breeding. Many researchers have mapped QTLs controlling shattering using genetic populations and linkage maps, with extensive studies in rice shattering and pod dehiscence in soybean and Arabidopsis, though research on shattering in Polygonaceae buckwheat species remains limited. Understanding QTLs for rice shattering, Arabidopsis pod dehiscence, and soybean pod shattering provides theoretical foundations for studying shattering/dehiscence in other plants.

4.3.1 Rice Most QTLs for rice shattering have been mapped to chromosomes 1, 3, 4, 5, 6, and 7, with none yet identified on chromosome 10. Genes on chromosome 1 include SH1, qSH1, and sh(t) (Zhu, 2008; Konishi et al, 2006; Zhu et al, 2014); on chromosome 4: SH4 (Li et al, 2006); on chromosome 6: shat and SH6(t) (Zhu et al, 2014; Zheng et al, 2016); and on chromosome 7: Oryza sativa CTD phosphatase-like1 (OsCPL1) (Ji et al, 2010). Although these genes are associated with shattering, their internal mechanisms differ. OsCPL1 encodes a CTD phosphatase domain and may block abscission layer differentiation by reducing phosphatase activity, resulting in non-shattering phenotypes. During rice grain maturation, the qSH1 gene promotes abscission layer formation at the base of the hull, facilitating easy shattering. Subsequent research revealed that qSH1 also affects abscission layer development, caused by a SNP (single nucleotide polymorphism) in a cis-acting element (T replacing G in the 5' upstream regulatory region at 12 kb) that alters expression of a downstream protein-coding gene (an ortholog of replumless), thereby inhibiting normal qSH1 expression and preventing hull base abscission layer formation, leading to loss of shattering phenotype.

Li et al (2006) identified SH4, a Myb3 transcription factor homolog similar to qSH1, where a nucleotide substitution (G replacing T) in the first exon causes a lysine-to-asparagine replacement in the Myb3 DNA-binding domain, preventing normal abscission layer development and resulting in loss of shattering phenotype. Genes controlling rice shattering work cooperatively. Zhou et al (2012) found that SHAT1 expression in abscission zones is positively regulated by SH4, while SHAT1 maintains SH4 expression in abscission zones. Downstream of SH4 and SHAT1, qSH1 maintains persistent expression of SHAT1 and SH4 in abscission zones, promoting abscission layer formation (Figure 3 [Figure 3: see original paper]). Ji et al (2010) discovered that the shattering gene SH-H en-

codes rice carboxy-terminal domain (CTD) phosphatase-like protein (OsCPL1), with functional inhibition after mutation leading to abscission layer formation and shattering phenotype. Recently, Htun et al (2014) and Inoue et al (2015) showed that qSH3 is more dependent, requiring cooperation with qSH1 and SH4 to promote abscission zone cell development, similar to how SH5 requires qSH1 assistance to promote abscission zone development. Therefore, abscission zone development and function involve precise multi-gene regulation, as illustrated in Figures 3 [Figure 3: see original paper] and 4 [Figure 4: see original paper].

4.3.2 Arabidopsis In Arabidopsis, BOP1/2 (BLADE ON PETIOLE1/2) encode transcription factors of the nonexpressor of pathogenesis-related genes 1 and control floral organ abscission zone formation. Other transcription factors AGL15 and AGL18 also affect floral organ abscission, with overexpression of both delaying floral organ shedding and senescence (Adamczyk et al, 2007; Fernandez et al, 2000). Recently, Cai & Lashbrook (2008) used microarray screening to demonstrate that overexpression of AtZFP2 (ZINC FINGER PROTEIN2) not only delays floral organ abscission but also affects flower morphology and fertility.

Notably, Arabidopsis pod dehiscence is also related to organ abscission, primarily involving MADS-box genes SHP1/2 (SHATTERPROOF1/2) that control pod abscission zone development and FUL (FRUITFULL) that determines pod development. These genes interact to regulate pod development. Ferrandiz et al (2000) proposed that FUL negatively regulates SHP to balance normal pod development. Subsequent studies revealed additional genes involved in pod abscission (separation layer) development (Figure 5 [Figure 5: see original paper]). FUL and RPL work together, while SH1, SH2, ALCATRAZ (ALC), and INDEHISCENT (IND) are expressed only in pod abscission zones and are also related to abscission layer formation, all being inhibited by FUL. REPLUMLESS (RPL) restricts SH1/2 expression in the replum. Recently, upstream genes of FUL, SHP, ALC, and IND–FILAMENTOUS FLOWER (FIL), YABBY3 (YAB3), and JAGGED (JAG)–were found to jointly promote their expression, while being feedback-inhibited by RPL. ASYMMETRIC LEAVES (AS) has different effects on pod development depending on expression levels and locations: overexpression in pods inhibits KNAT1/BP, while low-level expression in the replum reduces inhibition of KNAT1/BP and RPL, allowing coordinated action of KNAT1/BP and RPL to affect replum development (Figure 5 [Figure 5: see original paper]).

4.3.3 Soybean Soybean pod dehiscence resembles organ abscission, and its QTLs are equally important. Significant progress has been made in mapping soybean pod dehiscence QTLs. By 2014, Suzuki et al (2010), Dong et al (2014), and Funatsuki (2014) had reported pod dehiscence gene Pdh1 and five abscission zone formation genes SHAT1-5 (Figure 1 [Figure 1: see original paper]). The earliest pod dehiscence gene mapped to chromosome 16 was Pdh1, identified from a recombinant inbred line population derived from shattering-resistant

and susceptible varieties PI 416937. Gene Pdh1 (Gm16g25580) is located 20 kb upstream of a 47 kb genomic region (29,621-29,668 kb) and is responsible for soybean pod dehiscence. A single nucleotide mutation (A/T) causes premature stop codon appearance, regulating pod dehiscence magnitude. Pdh1 encodes a homologous protein highly expressed in lignin-rich sclerenchyma tissues within pod walls, thus regulating lignin synthesis and pod opening. Yamada et al (2009) mapped pod dehiscence-related QTLs near qPDH1 using two populations (shattering-resistant line Harosoy × susceptible line Toyomusume F2 and Kariyutaka × Wasekogane F2), explaining that although genetic backgrounds differ, QTLs also map near qPDH1, most likely because heterozygous parents produce offspring with high shattering traits, with shattering resistance showing near-recessive inheritance and major QTLs located at qPDH1.

Dong et al (2014) compared nucleotide genetic diversity between wild and cultivated soybeans, finding reduced nucleotide diversity in two genes, Gm04g39210 and Gm16g02200, during domestication, with no genetic variation in soybean germplasm at these candidate genes. Gm16g02200 overlaps with QTLs related to soybean pod dehiscence (Grant et al, 2009). Genetic and functional analyses revealed that Gm16g02200, named SHATTERING1-5 (SHAT1-5), is an ortholog of Arabidopsis AtNST1/2 encoding a NAC domain transcription factor (Table 2). Gene expression and transgenic complementation experiments confirmed that SHAT1-5 controls shattering-resistant phenotypes in cultivated soybeans by increasing its own expression in fiber cap cells (FCCs). SHAT1-5 and Pdh1 jointly regulate secondary cell wall thickening and shattering resistance in crop varieties (Dong et al, 2014).

4.3.4 Buckwheat In Polygonaceae buckwheat species, Yue et al (2012) successfully applied SSR markers to construct genetic maps and study germplasm resources and agronomic traits (including shattering) in common buckwheat. Genetic mapping showed that shattering genes Sht1 and Sht2 are tightly linked to markers S1182-1160 and S1182-1048, respectively. The molecular regulatory functions of Sht1 and Sht2 remain unclear, and other shattering genes have not been identified, indicating that research on shattering in buckwheat species is relatively limited and requires further effort.

4.4 Key Genes Regulating Flower and Fruit Abscission

Table 2 lists some genes related to flower and fruit abscission in plants. Genes affecting abscission can be broadly categorized into SHATTERING genes and MADS-box genes. SHATTERING genes are commonly found in food crops (rice, sorghum, maize, etc.) and model plants (Arabidopsis, etc.), primarily regulating abscission cell formation and development. MADS-box genes are widespread in legume plants such as soybean and Arabidopsis, regulating pod abscission cell lignification as transcription factors and thereby affecting abscission layer development, pod dehiscence, and flower/fruit abscission (Ferrandiz et al, 2000; Roberts et al, 2002; Pinyopich et al, 2003; Hofmann et al, 2012; Nakano et al,

2012; Dong et al, 2014). These genes are mainly divided into several families: the YABBY family affects plant development through multiple pathways (Lin et al, 2012; Sharma et al, 2014); the Myb3 family influences floral organ abscission development by encoding unknown transcription factors homologous to Myb3 (Li et al, 2006; Yan et al, 2015); the BEL1 family encodes BEL1 homeobox proteins affecting abscission development (Konishi et al, 2006); and the WRKY family encodes related proteins regulating abscission layer formation (Tang et al, 2013; Htun et al, 2014).

In most plants, genes function through regulatory networks, though some act independently. McKim et al (2008) and Chang et al (2015) found that the cotton GhBOP1 gene sequence shows high similarity to Arabidopsis BOP genes, with GhBOP1 predominantly expressed in cotton roots and controlling abscission zone cell differentiation. BOP1/2 are functionally redundant genes encoding transcription factors of nonexpressor of pathogenesis-related genes 1, present in most plant reproductive organs, especially during floral organ abscission zone development, though expressed differently in various plant abscission zones (Table 2). While BOP1/2 genes are redundant in Arabidopsis, cotton (GhBOP1), and tobacco (NtBOP2), they function independently during vegetative (petiole) and reproductive (inflorescence) development in barley (Jost et al, 2016).

4.5 Transcriptome Analysis of Abscission

Since the completion of Arabidopsis genome sequencing in 2000, sequence analyses of organ abscission or abscission zone-related genes have been conducted in Arabidopsis (Cho et al, 2000; Ogawa, 2009), rice (Li et al, 2006), soybean (Tucker et al, 2002), and other plants, with related gene sequences available in NCBI GeneBank. Recent research has focused on transcriptome sequencing of buckwheat inflorescences, fruits, leaves, and roots, identifying approximately 28 different gene sequences potentially related to pedicel abscission zone formation (Li et al, 2015; Huang et al, 2017). RNA-seq has also been applied to other plants. For example, in melon, the fruit pedicel abscission zone at early fruit ripening stages is downregulated by MAD-box, AP2/ERF, and Aux/IAA transcription factors but upregulated by Homeobox, Zinc finger, bZIP, and WRKY transcription factors. In later stages, the abscission zone is upregulated by MYB transcription factors, indicating different gene regulation between early and late stages. Gao et al (2016) constructed cDNA libraries from three rose petal abscission zone stages (GM1, GM3, GM5) and used Illumina sequencing technology to build corresponding transcript databases. Expression profiling of petal abscission zones identified 2,571 abscission-related transcripts and the abscission-responsive gene RhIAA16, which is expressed not only in abscission zones but also in other flower parts including petals, receptacles, stamens, and pistils, with higher expression in abscission zones and pistils. RhIAA16 shows highest expression before abscission (during abscission zone initiation) and decreases after initiation, demonstrating its involvement in abscission zone activation.

Research on flower and fruit abscission has primarily focused on model plant

Arabidopsis and staple crops rice, wheat, and sorghum (Meir et al, 2010; Li et al, 2006; Lin et al, 2012) and has expanded to other crops to address practical production issues such as buckwheat shattering (Chen et al, 2012), cotton boll shedding (Chang, 2016), and soybean pod dehiscence (Funatsuki et al, 2014). Previous research concentrated on physiological regulation by hormones and enzymes, while molecular mechanisms of abscission zone development and abscission remain relatively understudied. The relationship between full-length sequences of plant organ abscission genes and sequence variation is still unclear. Future research integrating transcriptome sequencing, full-length analysis of shattering-related genes, expression patterns, sequence variation, and morphological anatomy will elucidate variation patterns of shattering-related genes and their relationship with abscission layer formation. Molecular marker-assisted breeding will provide solutions to shattering problems and insights into plant origin and evolution. New advances in plant abscission molecular biology are expected in the near future, deepening our understanding of the fundamental mechanisms and contributing to modern agricultural production applications.

References

ADDICOTT FT, LYNCH RS, CARNS HR, 1955. Auxin gradient theory of abscission regulation[J]. Sci, 121(3148): 644-645.

ADAMCZYK BJ, LEHTI-SHIU MD, FERNANDEZ DE, 2007. The MADS domain factors AGL15 and AGL18 act redundantly as repressors of the floral transition in Arabidopsis[J]. Plant J, 50(6): 1007-1019.

ALONSO-CANTABRANA H, RIPOLL JJ, OCHANDO I, et al, 2007. Common regulatory networks in leaf and fruit patterning revealed by mutations in the Arabidopsis ASYMMETRIC LEAVES1 gene[J]. Dev, 134(14): 2663-2671.

ANTHONY MF, COGGINSJR CW, 1999. The efficacy of five forms of 2, 4-D in controlling preharvest fruit drop in citrus[J]. Sci Hortic, 81 (3): 267-277.

ARNAUD N, IRIN T, SOREFAN K, et al, 2010. Gibberellins control fruit patterning in Arabidopsis thaliana[J]. Genes Dev, 24(9): 2127-2132.

BAER J, TAYLOR I, WALKER JC, et al, 2016. Disrupting ER-associated protein degradation suppresses the abscission defect of a weak hae hsl2 mutant in Arabidopsis[J]. J Exp Bot, 67(18):

BAR-DROR T, DERMASTIA M, KLADNIK A, et al, 2011. Programmed cell death occurs asymmetrically during abscission in tomato[J]. Plant Cell, 23(4): 146-413.

BAIRD LM, REID MS, WEBSTER BD, 1984. Anatomical and physiological effects of silver thiosulfate on ethylene-induced abscission in Coleus[J]. J Plant Growth Regul, 3(1-4):

BELFIELD EJ, RUPERTI B, RORERTS JA, et al, 2005. Changes in expansin

activity and gene expression during ethylene promoted leaflet abscission in Sambucus nigra[J]. J Exp Bot, 56(413): 817-823.

BOWMAN JL, ESHED Y, BAUM SF, et al, 2002. Establishment of polarity in angiosperm lateral organs[J]. Trends Genetics, 18(3): 134-141.

BROWN KM, 1997. Ethylene and abscission[J]. Physiol Plantarum, 100(3): 567-576.

BRUMMELL DA, HALL BD, BENNETT AB, 1999. Antisense suppression of tomato endo-1,4-beta-glucanase Cel2 mRNA accumulation increases the force required to break fruit abscission zones but does not affect fruit softening[J]. Plant Mol Biol, 40(4): 615-622.

BURR CA, LESLLIE ME, RLOWSKI SK, et al, 2011. CAST AWAY, a membrane-associated receptor-like kinase, inhibits organ abscission in Arabidopsis[J]. Plant Physiol, 156(4):

CAI S, LASHBROOK CC, 2008. Stamen abscission zone transcriptome profiling reveals new candidates for abscission control: enhanced retention of floral organs in transgenic plants overexpressing Arabidopsis ZINC FINGER PROTEIN2[J]. Plant Physiol, 146(3): 1305-1321.

CAMPILLO ED, BENNETT AB, 1996. Pedicel breakstrength and cellulase gene expression during tomato flower abscission[J]. Plant Physiol, 111(3): 813-820.

CHANG J, JIN X, PANG JH, et al, 2015. Cloning of abscission zone development gene gossypiumhirsutum L. GhBOP1 and preliminary analysis of Its function[J]. Acta Agric Boreali-Sinica, 30(3): 14-19.

CHEN QF, YEN C, YANG JL, 1998. Chromosome location of the gene for brittle rachis in the Tibetan weedrace of common wheat[J]. Genetics Resour Crop Evol, 45(5): 407-410.

CHEN MK, HSH WH, LEE PF, et al, 2011. The MADS-box gene, FOREVER YOUNGFLOWER, acts as a repressor controlling floral organ senescence and abscission in Arabidopsis[J]. Plant J, 68(1): 168-185.

CHO HT, COSGROVE DJ, 2000. Altered expression of expansin modulates leaf growth and pedicel abscission in Arabidopsis thaliana[J]. Proc Natl Acad Sci USA, 97(17): 9783-9788.

DONG Y, WANG YZ, 2015. Seed SHATTERING: from models to crops[J]. Front Plant Sci, 6(476):

DONG Y, YANG X, LIU J, et al, 2014. Pod SHATTERING resistance associated with domestication is mediated by a NAC gene in soybean[J]. Nat Commun, 5(2): 1-11.

DOORN WGV, STEAD AD, 1997. Abscission of flowers and floral parts[J]. J Exp Bot, 48(4):

ELLIS CM, NAGPAL P, YOUNG JC, et al, 2005. AUXIN RESPONSE FACTOR1 and AUXIN RESPONSE FACTOR2 regulate senescence and floral organ abscission in Arabidopsis thaliana[J]. Deve, 132(20): 4563-4574.

EO J, LEE BY, 2009. Effects of ethylene, abscisic acid and auxin on fruit abscission in water dropwort (Oenanthe stolonifera, DC.)[J]. Sci Hortic, 123(2): 224-227.

FAETH SH, CONNOR EF, SIMBERLOFF D, 1981. Early leaf abscission: a neglected source of mortality for folivores[J]. Am Nat, 117(3): 409-415.

FERNANDEZ DE, HECK GR, PERRY S, et al, 2000. The embryo MADS domain factor AGL15 acts postembryonically. Inhibition of perianth senescence and abscission via constitutive expression[J]. The Plant Cell, 12(2): 183-198.

FUNATSUKI H, SUZUKI M, HIROSE A, et al, 2014. Molecular basis of a shattering resistance boosting global dissemination of soybean[J]. Proc Natl Acad Sci India, 111(50): 17797-17802.

GAO M, WANG X, WANG D, et al, 2009. Regulation of cell death and innate immunity by two receptor-like kinases in Arabidopsis[J]. Cell Host Micobe, 6(1): 34-44.

GAO Y, LIU C, LI X, et al, 2016. Transcriptome profiling of petal abscission zone and functional analysis of an Aux/IAA Family gene RhIAA16 involved in petal shedding in Rose[J]. Front Plant Sci, 7(106): 1375-1388.

GRAY WM, ESTELLE M, 2000. Function of the ubiquitin-proteasome pathway in auxin response[J]. Trends Bioch Sci, 25(3): 133-138.

GREB T, CLARENZ O, SCHAFER E, et al, 2003. Molecular analysis of the LATERAL SUPPRESSOR gene in Arabidopsis reveals a conserved control mechanism for axillary meristem formation[J]. Gene Dev, 17(9): 1175-1187.

GU Q, FERRANDIZ C, YANOFSKY MF, et al, 1998. The fruitfull MADS-box gene mediates cell differentiation during Arabidopsis fruit development[J]. Dev, 125(8): 1509-1517.

GUAN XX, 2015. Preliminary screening and functional identification of ARFS during tomato flower pedicel abscission[D]. Shenyang: Shenyang Agricultural University.

GUIBOILEAU A, SORMANI R, MEYER C, et al, 2010. Senescence and death of plant organs: nutrient recycling and developmental regulation[J]. C R Biol, 333(4): 382-391.

HADFIELD KA, BENNETT AB, 1998. Polygalacturonases: many genes in search of a function[J]. Plant Physiol, 117(2): 337-343.

HAGEN G, GUILFOYLE TJ, 2002. Auxin-responsive gene expression: genes, promoters and regulatory factors[J]. Plant Mol Biol, 49(3-4): 373-385.

HAN J, WANG YQ, 1999. Progress in researches on the mechanism of abscission of plant organs[J]. BullBot, 16(4): 405-410.

HTUN TM, INOUE C, CHHOURN O, et al, 2014. Effect of quantitative trait loci for seed shattering on abscission layer formation in Asian wild rice Oryza rufipogon[J]. Breeding Sci, 64 (3): 199-205.

HUANG J, DENG J, CHEN QF, 2017. Transcriptome analysis of Fagopyrum root and identification of genes involved in flavonoid biosynthesis[J]. J Agric Sci Tech-iran, 19(2): 9-19.

IGLESIAS DJ, LEVY Y, GOMEZCADENAS A, et al, 2004. Nitrate improves growth in salt-stressed citrus seedlings through effects on photosynthetic activity and chloride accumulation[J]. Tree Physiol, 24(9): 1027-1034.

INOUE C, HTUN TM, INOUE K, et al, 2015. Inhibition of abscission layer formation by an interaction of two seed-shattering loci, sh4 and qSH3, in rice[J]. Jpn J Gene, 90(1): 1-9.

ISHII T, NUMAGUCHI K, MIURA K, et al, 2013. OsLG1 regulates a closed panicle trait in domesticated rice[J]. Nat Genetics, 45(4): 462-465.

JI H, KIM SR, KIM YH, et al, 2010. Inactivation of the CTD phosphatase-like gene OsCPL1 enhances the development of the abscission layer and seed shattering in rice[J]. Plant J, 61(1):

JINN TL, STONE JM, WALKER JC, 2000. HAESA, an Arabidopsis leucine-rich repeat receptor kinase, controls floral organ abscission[J]. Gene Dev, 14(1): 108-17.

JOST M, TAKETA S, MASCHER M, et al, 2016. A Homolog of Blade-On-Petiole 1/2 (BOP1/2) controls internode length and homeotic changes of the barley inflorescence[J]. Plant Physiol, 171(2): 1113-1127.

KANDASAMY MK, MC KINNEY EC, DEAL RB, et al, 2005. Arabidopsis ARP7 is an essential actin-related protein required for normal embryogenesis, plant architecture, and floral organ abscission[J]. Plant Physiol, 138(4): 2019-2032.

KIM J, 2014. Four shades of detachment: Regulation of floral organ abscission[J]. Plant Signal Behav, 9(11): 40-45.

KIM J, PATTERSON SE, BINDBEUTEL RK, 2013. Floral organ abscission is regulated by the peptide ligand IDA and the receptor kinases HAE/HSL2[J]. Plant Physiol, 161(3): 1155-1167.

KONISHI S, IZAWA T, LIN SY, et al, 2006. An SNP caused loss of seed shattering during rice domestication[J]. Sci, 312(5778): 1392-1396.

KUMPF RP, SHI CL, LARRIEU A, 2013. Floral organ abscission peptide IDA and its HAE/HSL2 receptors control cell separation during lateral root emergence[J]. Proc Natl Acad Sci, 110(13):

LASHBROOK CC, GONZALEZ-BOSCH C, BENNETT AB, 1994. Two divergent endo-beta-1,4-glucanase genes exhibit overlapping expression in ripening fruit and abscising flowers[J]. Plant Cell, 6(10): 1485-1493.

LEHMANN K, HAUSE B, ALTMANN D, 2001. Tomato ribonuclease LX with the functional endoplasmic reticulum retention motif HDEF is expressed during programmed cell death processes including xylem differentiation, germination, and senescence[J]. Plant Physiol, 127(2): 436-449.

LERS A, KHALCHITSKI A, LOMANIEC E, et al, 1998. Senescence induced RNases in tomato[J]. Plant Mol Biol, 36(3): 439-449.

LERS A, SONEGO L, GREEN PJ, et al, 2006. Suppression of LX ribonuclease in tomato results in a delay of leaf senescence and abscission[J]. Plant Physiol, 142(2): 710-721.

LESLIE ME, LEWIS WE, YOUN JY, et al, 2009. The EVERSHED receptor-like kinase modulates floral organ shedding in Arabidopsis[J]. Dev, 331(2): 467-476.

LEWIS MW, LESLIE ME, FULCHER EH, et al, 2010. The SERK1 receptor-like kinase regulates organ separation in Arabidopsis flowers[J]. Plant J, 62(5): 817-828.

LI LF, OLSEN KM, 2016. To have and to hold: selection for seed and fruit retention during crop domestication[J]. Curr Top Dev Biology, 119: 63-109.

LI X, HUANG ZQ, JIANG J, et al, 2015. Screening of seed-holding gene in different Buckwheat varieties[J]. Guizhou Agric Sci, 43 (5): 19-23.

LI Z, MA J, SUI SZ, et al, 2014. Correlation research on the break strength, expansion protein activity and abscission layer growth of bud in wintersweet[J]. J Beijing For University,36(1): 109-113.

LILJEGREN SJ, LESLIE ME, DARNIELLE L, et al, 2009. Regulation of membrane trafficking and organ separation by the NEVERSHED ARF-GAP protein[J]. Dev, 136 (11) : 1909-1918.

LIN Z, LI X, SHANNON LM, YEH CT, et al, 2012. Parallel domestication of the SHATTERING1 genes in cereals[J]. Nat Genetics, 44(6): 720-724.

LU P, 2000. Chromosome karyotype analysis and location of the gene for brittle rachis in the Tibetan wheat[J]. Tibetan J Agric Sci, 22(2): 23-27.

MAO L, BEGUM D, CHUANG H, et al, 2000. JOINTLESS is a MADS-box gene controlling tomato flower abscission zone development[J]. Nature, 406(6798): 910-913.

MCMANUS MT, OSBROE DJ, 1990. Identification of Polypeptides specific to rachis abscission zone cells of Sambucus nigra[J]. Physiol Plant, 79(3): 471-478.

MCKIM SM, STENVIK GE, BUTENKO MA, et al, 2008. The BLADE-ON-PETIOLE genes are essential for abscission zone formation in Arabidopsis[J].

Dev, 135: 1537-1546.

MCWILLIAM JR, 1980. Development and significance of seed retention in grasses[A]. Seed production[C]. Hebblethwaite PD. Ltd London: Butter worth-Heinrman, 51-60.

MEIR S, PHILOSOPH-HADAS S, SUNDARESAN S, et al, 2010. Microarray analysis of the abscission related transcriptome in the tomato flower abscission zone in response to auxin depletion[J]. Plant Physiol, 154(4): 1929-1956.

MENG X, ZHOU J, TANG J, et al, 2016. Ligand-induced regulates receptor-like kinase complex floral organ abscission in Arabidopsis[J]. Cell Rep, 14(6): 1330-1338.

MICHELI F, 2001. Pectin methylesterases: cell wall enzymes with important roles in plant physiology[J]. Trends Plant Sci, 6(9): 414-419.

MOLINE HE, BOSTRACK JM, 1972. Anatomical and histochemical studies of abscission in coleus[J]. J Amer Soc Hort Sci, 97(1): 17-22.

NAKANO T, KIMBARA J, FUJISAWA M, et al, 2012. MACROCALYX and JOINTLESS interact in the transcriptional regulation of tomato fruit abscission zone development[J]. Plant Physiol, 158(1): 439-450.

OGAWA M, KAY P, WILSON S, et al, 2009. Arabidopsis dehiscence zone polygalacturonase1 (ADPG1), ADPG2, and QUARTET2 are polygalacturonases required for cell separation during reproductive development in Arabidopsis[J]. Plant Cell, 21(1): 216-33.

OKUSHIMA Y, MITINA I, QUACH HL, et al, 2005. Auxin response factor 2 (ARF)2: a pleiotropic developmental regulator[J]. Plant J, 43(1): 29-46.

OU YJ, JIANG JX, ZHANG TZ, et al, 2007. The function of Endo-1, 4- -glucanase on plant cell growth and development[J]. Acta Bot Boreal-Occident Sin, 27(4): 4844-4851.

PAN SJ, CHEN QF, 2010. Genetic mapping of common buckwheat using DNA, protein and morphological markers[J]. Hereditas, 147(1): 27-33.

PARRA-LOBATO MC, GOMEZ-JIMENEZ MC, 2011. Polyamine-induced modulation of genes involved in ethylene biosynthesis and signalling pathways and nitric oxide production during olive mature fruit abscission[J]. J Exp Bot, 62(13): 4447-4465.

PATHARKAR OR, WALKER JC, 2015. Floral organ abscission is regulated by a positive feedback loop[J]. Proc Natl Acad Sci, 112(9): 2906–2911.

PATHARKAR OR, WALKER JC, 2016. Core mechanisms regulating developmentally timed and environmentally triggered Abscission[J]. Plant Physiol, 172(1): 510-520.

PATTERSON SE, BLEECKER AB, 2004. Ethylene-dependent and independent processes associated with floral organ abscission in Arabidopsis[J]. Plant

Physiol, 134(1): 194-203.

PENG G, WU J, LU W, et al, 2013. A polygalacturonase gene clustered into clade E involved in lychee fruitlet abscission[J]. Sci Hortic, 150(2): 244-250.

PINYOPICH A, DITTA GS, SAVIDGE B, et al, 2003. Assessing the redundancy of MADS-box genes during carpel and ovule development[J]. Nature, 424(6944): 85-88.

PORTER KB, 1959. The inheritance of shattering in wheat[J]. Agron J, 51(3): 173-177.

ROBERTS JA, ELLIOTT KA, GONZALEZ-LARRANZA ZH, et al, 2002. Abscission, dehiscence and other cell separation processes[J]. Annu Rev Plant Biol, 53(1): 135-158.

ROLDAN MVG, PERILLEUX C, MORIN H, et al, 2017. Natural and induced loss of function mutations in SlMBP21 MADS-box gene led to jointless-2 phenotype in tomato[J]. Sci Rep, 7(1):

SAKAMOTO M, MUNEMURA I, TOMITA R, et al, 2008. Reactive oxygen species in leaf abscission signaling[J]. Plant Signal Behav, 3(11): 1014-1015.

SCHUMACHER K, SCHMITT T, ROSSBERG M, 1999. The lateral suppressor (LS) gene of tomato encodes a new member of the VHIID protein family[J]. Proc Natl Acad Sci USA, 96(1):

SEXTON R, ROBERTS JA, 1982. Cell biology of abscission[J]. Annu Rev Plant Biol, 33(33):

SIMONS KJ, FELLERS JP, TRICK HN, et al, 2006. Molecular characterization of the major wheat domestication gene Q[J]. Genetics, 172(1): 547-555.

SORMACHEVA I, GOLOVNINA K, VAVILOVA V, et al, 2015. Q gene variability in wheat species with different spike morphology[J]. Genet Resour Crop Ev, 62(6): 1-16.

STENVIK GE, TANDSTAD NM, GUO Y, et al, 2008. The EPIP peptide of inflorescence deficient in abscission is sufficient to induce abscission in Arabidopsis through the receptor-like kinases HAESA and HAESA-like2[J]. Plant Cell, 20(7): 1805-1817.

SUN FD, FENG NJ, ZHENG DF, et al, 2016. Effects of plant growth regulators S3307 and DTA-6 on physiological metabolism and GmAC of soybean pods[J]. Sci Agric Sin, 49 (4): 657-666.

SUNDARESAN S, PHILOSOPH-HADAS S, RIOV J, et al, 2015. Abscission of flowers and floral organs is closely associated with alkalization of the cytosol in abscission zone cells[J]. J Exp Bot, 66(5): 1355-1368.

SUZUKI M, FUJINO K, NAKAMOTO Y, et al, 2010. Fine mapping and development of DNA markers at the qPDH1 locus associated with pod dehiscence in soybean[J]. Mol Breeding, 25(3): 407-418.

TANG H, CUEVAS HE, DAS S, et al, 2013. Seed SHATTERING in a wild sorghum is conferred by a locus unrelated to domestication[J]. Proc Natl Acad Sci USA, 110(39): 15824-15829.

TANG L, CHEN QF, 2017. Study on the peduncle articulation anatomy of Fagopyrum plants[J]. Henan Agric Sci, 46(10) : 49-53.

TAYLOR JE, WHITELAW CA, 2001. Signals in abscission[J]. New Phytol, 151(2): 323-339.

TUCKER ML, WHITELAW CA, LYSSENKO NN, et al, 2002. Functional analysis of regulatory elements in the gene promoter for an abscission-specific cellulase from bean and isolation, expression, and binding affinity of three TGA-type basic leucine zipper transcription factors[J]. Plant Physiol, 130(3): 1487-1496.

ULMASOV T, HAGEN G, GUIFOYLE TJ, 1999a. Dimerization and DNA binding of auxin response factors[J]. Plant J, 19(3): 309-319.

WANG H, SUN X, YUE YL, et al, 2014. Association mapping of flower and pod abscission with SSR markers in northeast spring sowing soybeans[J]. Soil Crop, 3(1): 32-40.

WANG JE, QIU DF, ZHANG ZJ, 2017. The progress and prospect of rice shattering[J]. Hubei Agr Sci, 56(14): 2601-2604.

WANG XQ, XU WH, MA LG, et al, 2006. Requirement of KNAT1/BP for the development of abscission zones in Arabidopsis thaliana[J]. J Integr Plant Biol, 48(1): 15-26.

WANG YL, 2017. Study of molecular mechanism of MicroRNA1917 mediating tomato pedicel abscission[D]. Shenyang: Shenyang Agricultural University.

WANG M, YU Y, HABERER G, et al, 2014. The genome sequence of African rice (Oryza glaberrima) and evidence for independent domestication[J]. Nat Genetics, 46(9): 982-988.

WANG YJ, SCARTH R, CAMPBELL GC, et al, 2005. Inheritance of seed shattering in interspecific hybrids between Fagopyrum esculentum and F. homotropicum[J]. Crop Sci, 45(2):

WHITELAW CA, GONZALEZ-CARRANZA ZH, SWARUP R, 2002. Temporal and spatial expression of a polygalacturonase during leaf and flower abscission in oilseed rape and Arabidopsis[J]. Plant Physiol, 128(2): 534-543.

WU J, WANG F, CHENG L, et al, 2011. Identification, isolation and expression analysis of auxin response factor (ARF) genes in Solanum lycopersicum[J]. Plant Cell Rep, 30(11): 2059-73.

YAN H, MA L, WANG Z, et al, 2015. Multiple tissue-specific expression of rice seed-shattering gene SH4 regulated by its promoter pSH4[J]. Rice, 8(1): 12-21.

YAMADA T, FUNATSUKI H, HAGIHARA S, et al, 2009. A major QTL, qPDH1, is commonly involved in shattering resistance of soybean cultivars[J]. Breeding Sci, 59(4): 435-440.

YASUI Y, WANG YJ, OHNISHI O, et al, 1987. Construction of genetic maps of common buckwheat (Fagopyrum esculentum Moench) and its wild relative, F. homotropicum Ohnishi based on amplified fragment length polymorphism (AFLP) markers[J]. IntArch Allergy Imm, 83(3): 296-302.

YOON J, CHO LH, KIM SL, et al, 2014. The BEL1-type homeobox gene SH5 induces seed shattering by enhancing abscission-zone development and inhibiting lignin biosynthesis[J]. Plant J Cell & Mol Biol, 79(5): 717-728.

YUAN R, CARBAUGH DH, 2007. Effects of NAA, AVG, and 1-MCP on ethylene biosynthesis, preharvest fruit drop, fruit maturity, and quality of golden supreme and golden delicious apples[J]. J Am Soc Hortic Sci, 132(1): 101-105.

ZHANG L, 2013. Functional characterization of two shattering and yield related genes in common wheat (Triticum aestivum L.)[D]. Beijing: Chinese Academy of Agricultural Sciences Dissertation.

ZHANG Li, XUE JA, YU HQ, et al, 2012. The expression and function study of pectin methylesterase genes which regulate and control the petal falling in Arabidopsis[J]. J Plant Physiol, 48(4): 350-358.

ZHANG Z, BELCRAM H, GOTNICKI P, et al, 2011. Duplication and partitioning in evolution and function of homoeologous Q loci governing domestication characters in polyploid wheat[J]. Proc Natl Acad Sci USA, 108(46): 18737-18742.

ZHENG LY, WEI X, ZHOU K, et al, 2016. Identification of a rice chromosome segment substitution line Z481 carrying a major gene for seed shattering and mapping of SH6(t)[J]. Chin Sci Bull, 61(7): 748-758.

ZHOU Y, LU D, LI C, 2012. Genetic control of seed shattering in rice by the APETALA2 transcription factor shattering abortion1[J]. Plant Cell, 24(3): 1034-1048.

ZHU WY, ZHU Z, YANG DW, et al, 2008. Mapping of SH1, A dominant gene controlling seed shattering using SSR markers in rice (Oryza sativa L.)[J]. Chin Agric Sci Bull, 24(8): 84-87.

ZHU ZC, 2014. Genetic analysis and gene mapping of rice grain shattering in hybrid rice[J]. Hyb Rid Rice, 29 (1): 62-66.

Note: Figure translations are in progress. See original paper for figures.