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REVIEW

Gibberellin in plant height control: old player, new story

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Abstract Height relates to plant architecture, lodging resistance, and yield performance. Growth-promoting phytohormones gibberellins (GAs) play a pivotal role in plant height control. Mutations in GA biosynthesis, metabolism, and signaling cascades influence plant height. Moreover, GA interacts with other phytohormones in the modulation of plant height. Here, we first briefly describe the regulation of plant height by altered GA pathway. Then, we depict effects of the crosstalk between GA and other phytohormones on plant height. We also dissect the co-localization of GA pathway genes and established quantitative genetic loci for plant height. Finally, we suggest ways forward for the application of hormone GA knowledge in breeding of crops with plant height ideotypes.

Keywords Phytohormone · Gibberellin · Plant height · Interaction network · Quantitative genetic loci · Breeding application

Introduction

In nature, sessile plants establish their body plan in a fixed position. Unlike animals' hibernation and escape behavior, plants cannot evade unfavorable conditions. They have evolved intricate mechanisms to properly perceive, decode, and transmit the environmental signals. One strategy for environmental adaptation is the enhancement of plasticity and flexibility in plant architecture. The aerial and underground architecture of higher plants is determined by the shoot apical meristem (SAM) and root apical meristem (RAM), respectively. Plant growth is promoted by cell division and elongation of stem cells in the SAM and RAM. The SAM generates primary shoots, leaves, and floral organs. The axillary meristems (AMs) of shoots form a bud that is dormant or extends to generate a branch (Wang and Li 2008). The maintenance and modulation of SAM and AMs activities enable multiple dimensions of primary and lateral organs' growth and development, by which the diverse plant architecture is shaped.

Height, key component of plant architecture, has an important bearing on both natural beauty and yield performance. The increase of grain productivity during the 'Green revolution' mainly attributes to better agricultural practices and improved plant height. Plant height was improved by the introduction of semi-dwarf trait (Khush 2001). The semi-dwarf habit of high-yielding varieties developed during the 'Green revolution' is due to a significant change in plant hormone GA biosynthesis and signaling pathway (Hedden 2003). Additionally, crosstalk between GA and other phytohormones [auxin, brassinosteroids (BRs), ethylene (ET), jasmonates (JAs), and strigolactones (SLs)] functions in plant height control. During the past decades, roles of phytohormone GA in plant height determinant have been unraveled mainly through the study of GA deficiency and overdose mutants. In this review, we not only present the scenarios of GA mutants but also provide quantitative genetic cues on the function of GA in plant height modulation. We focus on cereal crop maize and extend to other plants, including *Arabidopsis*,

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rice, wheat, sorghum, and pea, when necessary. Information from this review will strengthen our understanding of molecular bases underlying plant height and provide guidance in breeding of crops with plant height ideotypes.

GA effects on plant height: mutant scenario

Diverse dwarf mutants related to GA biosynthesis, metabolism, and signaling have been identified. Systematical analysis of these dwarf mutants indicates that GA pathway change may lead to the altered plant height (Fig. 1). Maize dwarf mutants, *anther ear1* (*an1*) and *dwarf5* (*d5*), have defects in terpene synthase (TPS) enzymes involved in the early step of GA biosynthesis (Bensen et al. 1995; Fu et al. 2016). Similarly, mutation in any *Arabidopsis TPS* gene *GA1* or *GA2* results in the short stature (Sun and Kamiya 1994; Yamaguchi et al. 1998). Dwarf phenotypes of maize *dwarf3* (*d3*), *Arabidopsis ga3*, and rice *dwarf35* (*d35*) mutants attribute to lesions in P450 mono-oxygenases (P450s)-mediated steps of GA biosynthesis (Winkler and Helentjaris 1995; Helliwell et al. 1998; Itoh et al.

2004). The dwarfism phenotype is also observed in changes in 2-oxoglutaratedependent dioxygenases (2ODDs) enzymes GA 20-oxidase (GA20ox) and GA 3-oxidase (GA3ox) important for the final steps of GA biosynthesis. A deficiency in *Arabidopsis GA20ox* gene *GA5* causes the semi-dwarf phenotype (Xu et al. 1995). All mutations in *GA3ox* genes from three species, including *Dwarf1* (*D1*) from maize, *GA4* from *Arabidopsis*, and *Dwarf18* (*D18*) from rice, result in the dwarfism (Chiang et al. 1995; Itoh et al. 2001; Chen et al. 2014). Another 2ODDs enzyme GA 2-oxidases (GA2oxs) are responsible for GA turnover. Moreover, GA degradation could be achieved by ELONGATED UPPERMOST INTERNODE (EUI) protein through epoxidation. The internode length of recessive *eui* mutant is significantly increased (Zhu et al. 2006). Methylation modifications by GAMT1 and GAMT2 proteins also lead to GA deactivation. Intriguingly, gross morphology of single or double *GAMT1* and *GAMT2* mutants is similar to that of wild-type plants (Varbanova et al. 2007).

DELLA proteins are hub repressors of GA signaling. Gain-of-function mutation in *Arabidopsis DELLA* gene
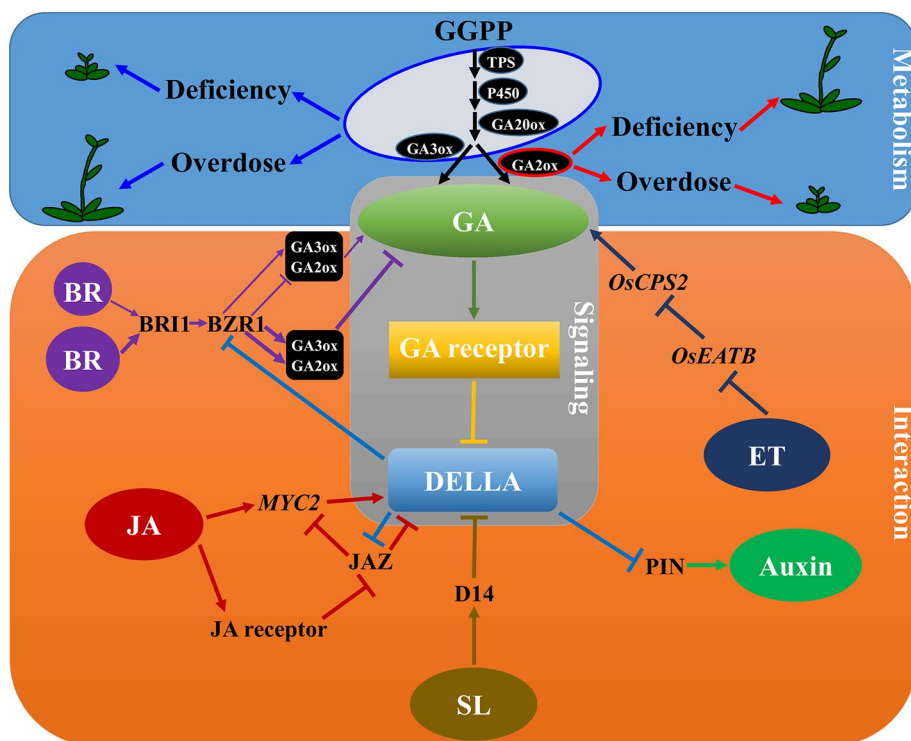
Fig. 1 Metabolism, signaling, and interaction atlas of plant hormone GA. For GA metabolism, bioactive GAs are synthesized from precursor GGPP catalyzed by three types of enzymes, including TPSs, P450s, and 2ODDs. Mutations in genes encoding GA biosynthetic and metabolic enzymes influence plant height. Phytohormone GA, GA receptor, and DELLA repressor shape a GA–GID1–DELLA regulatory module in the GA-signaling cascade. GA interacts with other phytohormones (auxin, BRs, ET, JAs, and SLs) in diverse biological processes, including cell elongation, and plant height modulation.

BRI1 BRASSINOSTEROID-INSENSITIVE1, *BZR1* BRASSINAZOLE RESISTANT1, *D14* DWARF14, *GGPP trans*-geranylgeranyl diphosphate, *JAZ* JA ZIM-domain, *OsEATB* rice ERF protein associated with tillering and panicle branching, *PIN* PIN-FORMED. *BR circled by relatively small round* represents normal physiological condition, and *BR circled by relatively large round* represents BR overdose. *Arrows* indicate positive regulation, and *T bars* denote negative regulation

gibberellin insensitive (*gai*) leads to the dwarf phenotype (Peng and Harberd 1993). Loss-of-function mutation in rice *DELLA* gene *Slender rice 1* (*SLR1*) causes dwarfism (Ikeda et al. 2001). In rice, F-box protein GIBBERELLIN-INSENSITIVE DWARF2 (GID2) is involved in the formation of the Skp–Cullin–F-box (SCF) E3 ubiquitin ligase complex that polyubiquitinates DELLA protein for its subsequent degradation by the proteasome. *gid2* mutant exhibits a GA-insensitive dwarf phenotype (Sasaki et al. 2003).

GA effects on plant height: phytohormone interaction scenario

The synergy and antagonism between GA and other phytohormones (auxin, BRs, ET, JAs, and SLs) play essential roles in plant height control (Fig. 1).

To coordinate GA metabolism during organ elongation, auxin-responsive auxin response factor (ARF) and auxin/indole-3-acetic acid (Aux/IAA) family members directly up-regulate the expression of multiple GA catabolic genes *GA20ox* and *GA2ox* (Frigerio et al. 2006). Additionally, the convergence of auxin and GA cascades can be mediated by PIN-FORMED (PIN) proteins, auxin efflux carriers. In GA-deficient mutant, auxin transport is significantly inhibited because of a degradation of PIN proteins induced by GA deficiency. The reduced auxin transport influences cotyledon differentiation and root gravitropism (Willige et al. 2011). There has been evidence supporting the relationship between auxin transport defects and dwarf phenotypes of maize *brachytic2* (*br2*) and sorghum *dwarf3* (*dw3*) mutants (Multani et al. 2003). It invites the question of whether PIN-mediated auxin–GA interplay functions in plant height control.

Plant hormones, GA and BR, have distinct but also overlapping roles in the regulation of organ elongation and ultimate plant height (Singh and Savaldi-Goldstein 2015). GA and BR pathways are integrated through the crosstalk between BRASSINAZOLE RESISTANT1 (BZR1), transcriptional modulator of the expression of BR-responsive genes, and DELLA protein, negative regulator of GA signaling. DELLA proteins repress regulatory activities of BZR1 by the inhibition of BZR1 binding to the promoters of target genes (Gallego-Bartolomé et al. 2012). In rice, BR triggers growth responses by the modulation of GA homeostasis. For one thing, physiological concentrations of BR enhance GA abundance by the increased expression of GA biosynthetic gene *GA3ox* to promote growth. For another, BR overdose represses GA biosynthesis and induces GA degradation through up-regulation of GA metabolic gene *GA2ox* to inhibit cell elongation (Tong et al. 2014). By contrast, BR content and response changes in pea are not accompanied by the altered GA levels (Jager et al. 2005). The interplay between GA and BR pathways may be species specific. The GA–BR interaction need be surveyed in the context of specific species, developmental stages, and physiological condition.

The crosstalk between GA and ET occurs under abiotic stress conditions. Member of APETALA2 (AP2)/ethylene-responsive factor (ERF) family *OsEATB* down-regulates the expression of GA biosynthetic gene *OsCPS2* to suppress ET-induced GA responses in rice, which leads to the inhibition of internode elongation and reduced plant height (Qi et al. 2011). During rice prolonged submergence, transcriptional regulators AP2/ERFs restrain GA biosynthesis and shoot elongation by up-regulation of GA catabolic gene *GA2ox* (Jung et al. 2010). Phytohormone ET interacts with GA to achieve the tradeoffs between plant growth and defense in adaption to abiotic stress.

GA and JA act antagonistically to modulate seedling growth during plant defense. In JA signaling, JA ZIM-domain (JAZ) repressors sequester MYC2, an important activator of JA responses. JA induces JAZs degradation and delays the degradation of DELLA proteins. The accumulation of DELLA proteins in turn represses JAZs and inhibits GA-promoted shoot elongation, which is beneficial to the release of activator MYC2 and the enhancement of JA-mediated defense responses (Hou et al. 2010; Wild et al. 2012; Yang et al. 2012). Similar to GA–ET interplay, the crosstalk between GA and JA confers resistance through the modulation of plant growth.

A novel class of terpenoid lactones SLs influence plant architecture, including shoot branching and plant height (Al-Babili and Bouwmeester 2015). In rice, the interaction between SL receptor DWARF14 (D14) and GA signaling repressor SLR1 has been reported, suggesting that SL and GA pathways can be integrated by the formation of D14–SLR1 complex (Nakamura et al. 2013). Although the interaction between SL receptor D14 and rice DELLA protein SLR1 has been confirmed, SLs and GAs could function independently during shoot elongation. For example, SLs promote internode elongation of pea shoot independently of GAs (de Saint Germain et al. 2013). GAs are also essential for shoot elongation. More evidence need be collected to unveil the exact role of GA–SL crosstalk in organ elongation and final plant height.

GA effects on plant height: quantitative genetics scenario

Understanding the genetic bases of plant height is not only beneficial to addressing fundamental issues in plant developmental biology but also helpful for the improvement of plant architecture in breeding programs. A number of efforts have been made to elucidate the genetic architecture of plant height. Height in some plants, such as maize and rice, has a quantitative nature (Wang and Li 2005;

Peiffer et al. 2014). Quantitative genetics approaches, including linkage mapping and genome-wide association study (GWAS), have been employed to identify quantitative genetic loci for plant height.

Classical linkage mapping or QTL mapping method based on bi-parental crosses is used to reveal genetic loci influencing plant height. Over 200 QTL for maize plant height have been deposited in the MaizeGDB reservoir (Andorf et al. 2016). However, identification of candidate genes underlying these QTL for maize plant height is deemed to be a daunting task mainly because of relatively large confidence interval. Meta-analysis has been proven to be an effective approach to refine QTL interval. We meta-analyzed of QTL for maize plant height. Results indicate that some GA pathway-related genes, *GA3ox2* and *GA2ox*, co-localize with established meta-QTL intervals (Wang et al. 2016b). Intriguingly, the aforementioned *GA3ox2* in meta-QTL interval is the gene within maize plant height QTL *qPH3.1* that has been map-based cloned (Teng et al. 2013). Of note, GA-deficient dwarf mutant *dwarf1* (*d1*) analysis demonstrates that maize *D1* corresponds to *GA3ox2* (Chen et al. 2014). Leveraging the separate QTL mapping, integrative QTL meta-analysis, and independent mutant survey information provides new insights into the role of phytohormone GA in plant height determinant.

GWAS or association mapping based on natural populations is employed to dissect the genetic architecture of plant height. Joint linkage–linkage disequilibrium mapping identifies numerous loci influencing maize plant height. Significant association revealed by GWAS co-localizes with putative GA receptor GID1L2 (Peiffer et al. 2014). GWAS analysis of maize plant height in another separate panel identifies association near *GA20ox* gene essential for the conversion of precursor GA_{12} to bioactive GAs (Weng et al. 2011). In wheat, GA-related genes within loci for plant height have been confirmed by integrated GWAS and syntenic analysis (Zanke et al. 2014). With marker saturation, phenome advance, and algorithm optimization, novel GA pathway effectors influencing plant height are likely to be unraveled by the GWAS approach.

It should be noted that the genetic architecture of plant height in sorghum is different from that in maize. As mentioned above, maize plant height is controlled by numerous loci with minor effects. Moreover, GA pathway-related genes, including *GA20ox, GA3ox, GA2ox*, and GA receptor gene, are the candidates underpinning quantitative genetic loci for maize plant height (Weng et al. 2011; Teng et al. 2013; Peiffer et al. 2014). In contrast, there are four major loci (*DW1, DW2, DW3*, and *DW4*) influencing sorghum plant height (Quinby and Karper 1954). These four major loci for sorghum plant height have been further confirmed by the GWAS results (Morris et al. 2013). Intriguingly, there are not GA pathway-related genes within four

sorghum plant height loci *DW1–DW4*. Recent GA-related mutant analysis indicates that GA-deficient dwarf phenotypes tend to pleiotropically induce culm bending in sorghum. GA-related dwarf traits are not likely to be selected during sorghum breeding due to the close relationship between GA-deficient dwarf phenotypes and negative pleiotropic effects (Ordonio et al. 2014). It is also the reason why none of GA pathway-related genes have been discovered within major loci influencing sorghum plant height.

GA effects on plant height: breeding application scenario

Efficient and sustainable increase of crop yield is an important priority in a changing world (Tester and Langridge 2010). Modification of plant height by the altered GA pathway has been proven to be effective for the improvement of crop yield performance. A landmark example is the development of high-yielding varieties during the 'Green revolution'. These high-yielding varieties are characteristic of high capacities of nitrogen assimilation and lodging resistance, which mainly attributes to the introduction of semi-dwarf traits. The semi-dwarf habit is due to GA pathway change that is governed by GA biosynthetic gene *sd1* in rice and GA signaling gene *Rht* in wheat (Peng et al. 1999; Monna et al. 2002; Spielmeyer et al. 2002).

With rapid advances in omics-based technologies and computational toolkit, the development of varieties with plant height ideotypes through GA pathway change can be illuminated at multiple layers (Fig. 2). For one thing, dwarf germplasm is collected and grouped into GA-sensitive and GA-insensitive types based on their GA responses. GA mutants with mild mutation effects and breeding potential can be directly adopted in breeding practice. For another, genes underlying dwarf phenotypes due to GA defects can be isolated by transposon tagging and map-based cloning methods. Cloned genes are informative for the identification, transfer, and pyramiding of elite allele. Moreover, interaction network of GA and other phytohormones can be elucidated through systems biology approaches, such as co-expression analysis and meta-analysis of hormone transcriptome (Brenner et al. 2012; Saithong et al. 2015). Hub regulators of GA-involved regulatory network in plant height control can be discovered. In addition, GA-related effectors of plant height control can be dissected by linkage mapping and GWAS approaches. The breeding-favored effectors can be recruited for the improvement and enhancement of germplasm bank specifically tailored to dwarf varieties breeding.

GA-deficient dwarf mutants tend to exhibit negative pleiotropic effects, such as severely shortened internodes and degenerated inflorescence organs (Wang et al. 2016a). Breeding potential of severe GA-deficient mutants seems
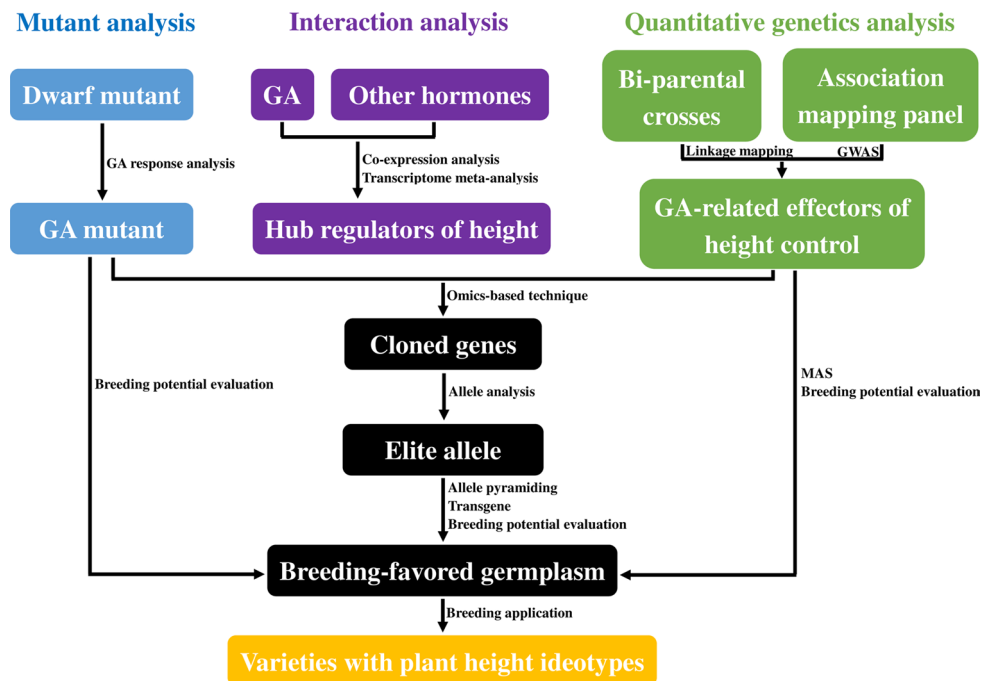
Fig. 2 Breeding of crops with plant height ideotypes by altered GA pathway. GA dwarf mutants with mild mutation effects and breeding potential can be directly used in breeding practice. Causative genes underlying GA dwarf phenotypes are identified by transposon tagging and map-based cloning methods. Systems biology approaches, such as co-expression analysis and meta-analysis of hormone transcriptome, are employed for the dissection of hub regulators of GA-mediated network of plant height determinant. GA-related effectors of plant height control can be discovered by linkage mapping and GWAS approaches. To develop crops with plant height ideotypes, isolated causative genes, hub regulators, and effectors are employed for the identification, transfer, and pyramiding of elite allele through MAS and transgene strategies. *GWAS* genome-wide association study, *MAS* marker-assisted selection

marginal. In such case, novel GA-deficient plants with mild mutation effects can be identified through the survey of geographically diverse germplasm. Alternatively, some biotechnological methods, including marker-assisted selection (MAS) and transgene can be used to modify plant height through the regulation of GA pathway (Fig. 2). The advent of CRISPR/Cas9 genome-editing platforms will facilitate the genetic modulation of plant height by altered GA pathway. Editing of *Arabidopsis* GA signaling gene *GAI* with the CRISPR/Cas9 system results in a dwarf phenotype (Feng et al. 2013). One of the keys to reshaping plant height via genetic interference of GA pathway is the selection of suitable genes that positively affect height without penalties on yield and other agronomic traits. Some GA pathway genes belong to gene family members, such as *GA20oxs* and *GA3oxs*. The expression of *GA20oxs* and *GA3oxs* genes can be finely tuned, which is beneficial to the avoidance of undesirable pleiotropy. *GA20oxs* and *GA3oxs* could be prioritized for the improvement of plant height.

GA pathway change not only affects plant height but also influences biotic and abiotic stress tolerance, floral organ morphogenesis and fertility, and quality traits (Achard et al. 2008; Plackett et al. 2012; Saville et al. 2012;

Voorend et al. 2016; Wu et al. 2016). In the breeding relevant context, diverse roles of GA can be a two-edged sword. For one thing, dwarf germplasm with unfavorable pleiotropy due to GA deficiency or overdose may limit its breeding application. For another, functional diversity of phytohormone GA holds promise for improved plant architecture, stress tolerance, fertility, quality, and yield benefits through the modification of GA cascade.

Conclusion

Height, key component of plant architecture, is related to lodging resistance, photosynthetic capacity, harvest index, and ultimate yield performance. Ample evidence supports the role of phytohormone GAs in plant height control. Systematic characterization of GA pathway mutants in diverse species indicates that changes in the biosynthesis, metabolism, and signaling cascades of GA influence plant height. Some GA pathway genes belong to gene family members, such as *GA20oxs, GA3oxs*, and *GA2oxs*. Deficient in one member of *GA20oxs, GA3oxs*, and *GA2oxs* may not result in the altered phenotype due to functional redundancy. In such case, double, triple,

and quadruple mutant populations need be developed to untangle their exact roles and the combinatorial interplay among *GA20oxs, GA3oxs*, and *GA2oxs* in plant height control. GA2ox enzymes are responsible for GA degradation. GA turnover can also be achieved through epoxidation and methylation modifications. Novel mechanisms of GA degradation may be discovered by forward and reverse genetic approaches. Interaction between GA and other phytohormones adds the complexity of GA-mediated regulatory network of plant height. For example, GA–BR interaction varies among species, developmental stages, and physiological condition. In such case, function of the interplay between GA and other phytohormones in plant height modulation need be surveyed in species-specific, developmental, and physiological contexts. In addition, regulatory hubs and edges of GA-mediated network could be identified through the systems biology methods, such as co-expression analysis and meta-analysis of hormone transcriptome. In some plants, plant height is quantitative, meaning that it is controlled by numerous loci with minor effects. Two complementary approaches, linkage mapping and GWAS, have been employed to identify loci for plant height. GA pathway genes co-localize with identified loci for plant height. Intriguingly, roles of some GA pathway genes in plant height have been confirmed by the mutant research and QTL meta-analysis. The integration of QTL mapping, QTL meta-analysis, and mutant survey provides complementary information for the understanding of the function of GA in plant height determinant. Moreover, combined linkage mapping and GWAS is helpful to the discovery of novel GA pathway genes relevant for plant height control. During the 'Green revolution', high-yielding varieties were developed by the introduction of semi-dwarf traits. The semi-dwarf habit mainly attributes to the altered GA pathway. However, the GA dwarf mutants tend to exhibit negative pleiotropic effects, which limits their breeding application. Thus, novel GA mutants with mild mutation effects and breeding potential need be isolated through the survey of geographically diverse germplasm. Moreover, diverse omics-based methods could be used to identify GA effectors for plant height control. Identified GA effectors can be engineered by some genome-editing techniques, such as CRISPR/Cas9 to reshape plant height. Additionally, isolated GA effectors could be served for the identification, transfer, and pyramiding of elite allele to boost crop productivity and food security, which is an important priority to cope with a constantly changing world.

Author contribution statement YW conceived the topic. JZ, WL, and DD collected background information. JZ and WL contributed to the figures. YW wrote the manuscript.

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