Tansley review

Genes for control of plant stature and form

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Contents

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Summary

Here we summarize progress in identification of three classes of genes useful for control of plant architecture: those affecting hormone metabolism and signaling; transcription and other regulatory factors; and the cell cycle. We focus on strong modifiers of stature and form that may be useful for directed modification of plant architecture, rather than the detailed mechanisms of gene action. Gibberellin (GA) metabolic and response genes are particularly attractive targets for manipulation because many act in a dose-dependent manner; similar phenotypic effects can be readily achieved in heterologous species; and induced pleiotropic effects – such as on nitrogen assimilation, photosynthesis, and lateral root production – are usually positive with respect to crop performance. Genes encoding transcription factors represent strong candidates for manipulation of plant architecture. For example, *AINTEGUMENTA*, *ARGOS* (auxin-regulated gene controlling organ size), and growth-regulating factors (*GRF*s) are strong modifiers of leaf and/or flower size. Plants overexpressing these genes had increased organ size and did not display negative pleiotropic effects in glasshouse environments. TCP-domain genes such as *CINCINNATA,* and the associated regulatory miRNAs such as *miRJAW,* may provide useful means to modulate leaf curvature and other foliage properties. There are considerable opportunities for comparative and translational genomics in nonmodel plant systems.

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I. Introduction

Plants show extensive and complex variations in stature and form. 'Stature' refers to overall size; height, tallness, and size are common synonyms. 'Form' is a much more vague term, and has diverse connotations that depend on context. Roget's Thesaurus (2007) has six senses as a noun and four senses as a verb. As a noun with the sense of 'shape', common synonyms include anatomy, appearance, architecture, design, model, and structure. Both stature and form show plasticity, which is itself widely known to vary among genotypes and species. However, consideration of genotype × environment dimensions of the control of plant stature and form are beyond the scope of this paper. We refer to form as any changes in the overt appearance of whole plants or plant organs, particularly in branching structure and leaf morphology.

Size tends to be far more plastic than form. Overall size can vary over five orders of magnitude, yet most aspects of plant form are remarkably consistent within species. For example, leaf size can vary as much as a thousandfold among species (e.g. < 0.1 cm^2 in *Lemna* to > 10 000 cm^2 in *Victoria*), yet at a similar developmental stage and environment it often varies by only a few per cent within a species (Mizukami, 2001). The low intraspecific variation of final size and shape suggests that there are fundamental developmental constraints on leaf form imposed by natural selection for adaptation and survival, and thus tight genetic controls during development (Mizukami, 2001; Weiss *et al.*, 2005). In this review, we focus on advances in understanding of the genetic bases of both size and form of plants and plant organs, with an emphasis on genes that can be used as tools for control of plant architecture. We discuss three classes of genes in depth: those affecting hormone metabolism and signaling; transcription and other regulatory factors; and the cell cycle. Other recent reviews have covered regulation of stature or organ size from developmental or gene regulation perspectives (Thomas & Sun, 2004; McSteen & Leyser, 2005; Schmitz & Theres, 2005; Vert *et al.*, 2005; Woodward & Bartel, 2005; Golz, 2006; Inze & Veylder, 2006; Anastasiou & Lenhard, 2007).

The introduction of the 'Green Revolution' dwarf and semi-dwarf varieties of wheat (*Triticum aestivum*) and rice (*Oryza sativa*) demonstrated the value of stature control. Wild-type cereals grow tall in response to high density, high fertility, and irrigation. They also make a proportionally larger in investment in foliage over grain biomass, reducing harvest index. The semi-dwarf varieties were shorter and more resistant to damage by wind and rain (lodging), and also responded better to nitrogen fertilizers by increasing grain yield rather than straw biomass. As a result, these varieties contributed substantially to dramatic increases in cereal crop yields world-wide (David & Otsuka, 1994).

In fruit trees, dwarf and semi-dwarf varieties are preferred for rootstocks or direct planting (Webster, 2002). These cultivars allow dense field cultivation; facilitate mechanized maintenance; increase efficiency of fruit collection; and allow more precise pesticide application, reducing spray drift (Webster, 2002). Because of their large size, trees require intensive maintenance to avoid damage to homes and power lines, and tree maintenance costs comprise a significant proportion of electrical utility budgets. Utility companies in the USA spend ~$1.5 billion per year trimming trees and controlling brush, including herbicide and growth-retardant treatments (EPRI, 1995). In spite of these expenditures, trees are the largest single cause of power outages (Simpson & Bossuyt, 1996). Pruning and tree removal are two of the highest street-tree maintenance costs; approx. 58% of urban tree-care budgets are allocated to tree trimming, removal, and disposal (Nowak, 1990). Trees that are intensively cultured as wood fiber crops may also be improved by semi-dwarfism and related alterations in form (Bradshaw & Strauss, 2001; Ragauskas *et al.*, 2006). Domesticated trees that are substantially shorter and stouter may produce less reaction wood, which degrades wood and pulp quality, in reaction to bending; give a higher harvest index; have improved harvesting/handling efficiencies; and enable greater unit-area fiber yields. Crowns of dwarfed trees will likely be narrower, an ideotype that should allow for a greater number of stems per unit area.

Final plant size and form are determined by the cell number and cell size resulting from post-embryonic cell division, expansion, and differentiation (Mizukami, 2001; Weiss *et al.*, 2005). Early studies, which were later substantiated by detailed molecular experiments, suggested that there is an intrinsic mechanism for coordination of cell division and expansion to produce a developmentally predefined 'normal' species size and form. The genes that encode plant hormones and their signaling clearly play a major role in regulation of these mechanisms.

II. Hormonal control

Plant hormones are major regulators of growth and development, and have dramatic effects on stature, form, and physiology. Early experiments with exogenous applications pointed to roles in regulation of elongation growth, flowering, apical dominance, lateral/adventitious root formation, and vascular differentiation (Davies, 1995). Some of these applications have been commercialized and provided important improvements in crop propagation and management (Woodward & Bartel, 2005). More recently, genetic dissection has allowed new insights into the molecular mechanism of hormone biosynthesis and signal transduction pathways, and has provided new options for crop improvement (Sakamoto, 2006). We review the effect of auxin, gibberellins, brassinosteriods, and a novel hormone in regulation of plant form and size, but ignore the roles of cytokinins (CKs) and ethylene. They can also modulate plant growth responses, but because their effects are frequently less specific, they are more prone to have undesirable pleoiotropic effects, limiting their value for manipulation of plant stature and form.

1. Auxin – the master switch

There is probably not a single aspect of the growth and development of a plant that is not affected by auxin (Davies, 1995; Leyser & Berleth, 1999). The multiplicity of auxin responses reflects the central role that this hormone plays in coordinating growth and developmental effects in plants, and thus it is not surprising that genes involved in auxin biosynthesis and signal transduction can be strong modifiers of plant size and form. Auxin metabolism and signaling have been the subjects of extensive genetic, genomic, and biochemical dissection (reviewed in Abel & Theologis, 1996; Friml & Palme, 2002; Leyser, 2002; Liscum & Reed, 2002; Woodward & Bartel, 2005; Tanaka *et al.*, 2006; Teale *et al.*, 2006; Berleth *et al.*, 2007; DeSmet & Jurgens, 2007; Kerr & Bennett, 2007). Below we summarize the genes that are strong modifiers of plant form and shape.

Polar auxin transport Because auxin acts in a concentration-dependent manner and auxin gradients serve as positional signals, plants have developed an intricate system of auxin carriers that regulate hormone distribution (Friml & Palme, 2002). Efflux/influx carriers are transmembrane proteins that mediate the passage and residence time of auxin in cells (Liu *et al.*, 1993; Galweiler *et al.*, 1998; Muller *et al.*, 1998; Marchant *et al.*, 2002). In Arabidopsis, auxin influx is carried out by Auxin Permease1 (AUX1) and three LAX (like AUX1) proteins (Bennett *et al.*, 1996; Parry *et al.*, 2001). The efflux is mediated by PIN-FORMED (PIN) proteins, encoded by a gene family of eight members (Galweiler *et al.*, 1998; Muller *et al.*, 1998; Friml & Palme, 2002; Friml *et al.*, 2002, 2003).

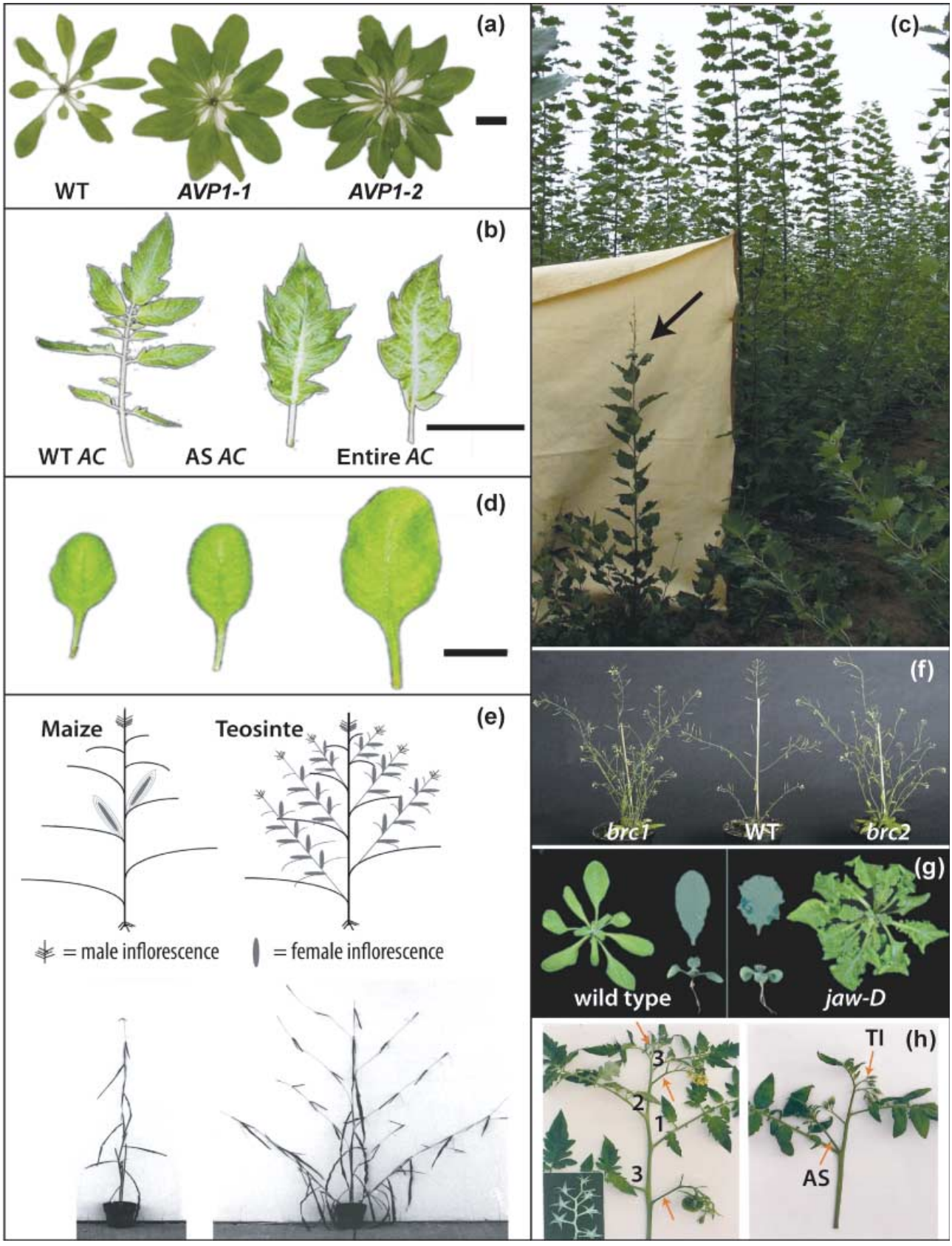
Recently, auxin carriers have been implicated in controlling phyllotaxis, a major determinant of plant architecture (Reinhardt *et al.*, 2003; Jonsson *et al.*, 2006). Phyllotaxis is the periodic arrangement of leaves and branches along the stem that is characterized by Fibonacci numbers (Roberts, 1978). Through elegant expression analyses and micro-scale auxin applications, Reinhardt *et al.* (2003) provided evidence for the involvement of auxin carriers in the control of phyllotaxis. According to the model proposed, acropetal flux toward the apex is mediated by *PIN1* expression and intracellular redistribution, and creates regional sinks and high-concentration pockets of auxin that determine the periodicity of leaf emergence along the stem.

In addition to the AUX1 and PIN1 proteins, auxin transport is mediated by a group of ATP binding cassette (ABC) transporter proteins that show a high level of similarity to mammalian multi-drug-resistant genes, which are a subset of the P-glycoprotein (*PGP*) gene family (Noh *et al.*, 2001, 2003). To date, three PGP proteins in Arabidopsis have been found to mediate auxin transport (e.g. AtPGP1, 4, and 19) and loss-of-function mutations produce highly pleitropic auxin-related morphological abnormalities in roots and shoots (Noh *et al.*, 2001, 2003; Geisler *et al.*, 2004; Terasaka *et al.*, 2005; Bouchard *et al.*, 2006). The lesion in a gene encoding a similar PGP protein in sorghum (*Sorghum* spp.) and maize (*Zea mays*) seemed to condition more moderate phenotypes (Multani *et al.*, 2003; Salamini, 2003). Plants were characterized by compact lower stalk internodes, and no other plant organ was affected in size or growth. The mutant phenotype provided major agronomic benefits that, although unexploited in maize, are widely used in sorghum breeding (Multani *et al.*, 2003). Using transposon tagging, the gene was found to encode a PGP protein with high similarity to Arabidopsis AtPGP1 (Multani *et al.*, 2003).

Polar auxin transport is motivated by gradients in cytoplasmic and apoplastic pH (Grebe, 2005) that are set up by proton pump-transporter proteins. The acid theory of growth postulates that auxin-stimulated excretion of protons into the cell wall that causes acidification and loosening of the cell wall allows expansion. However, until lately there had been little molecular genetic evidence to support this theory. The recently cloned H^+ pyrophosphatase (PPase) *Arabidopsis vacuolar pyrophosphatase1* (*AVP1*) provides supporting evidence for this hypothesis (Grebe, 2005; Li *et al.*, 2005b). In addition to maintaining vacuolar pH, AVP1 was found to be localized in the plasma membrane, and its overexpression increased auxin transport, and loss of function decreased transport. Particularly interesting were the overexpression phenotypes, where ectopic expression of the protein increased the number of rosette leaves and leaf size, mainly via increased cell numbers (Fig. 1a). Overexpression was accompanied by a similar increase in root size.

***Aux/IAA* gene family** One of the hallmarks of auxin response in plants is the strong and rapid induction of *auxin/indole acetic acid* (*Aux/IAA*) genes (Abel & Theologis, 1996). They are primary auxin-response genes, meaning that their activation does not need *de novo* protein synthesis. Arabidopsis has 29 *Aux/IAA* genes, and mutant screens have identified mutations with distinct phenotypes in 10 of them (Liscum & Reed, 2002). The isolated mutations are predominantly gain-of-function lesions in the conserved domain II that is present in all gene family members; the mutations seem to render the protein resistant to degradation, and several size and form characteristics were found to be modified in the mutant plants. For example, *iaa3/short hypocotyl (shy)2-2* (Tian & Reed, 1999; Tian *et al.*, 2002), *iaa6/shy1-1* (Kim *et al.*, 1996), *iaa7/auxin resistant (axr)2-1* (Timpte *et al.*, 1994; Nagpal *et al.*, 2000), and *iaa17/axr3-1* (Rouse *et al.*, 1998) seem to condition shorter hypocotyls. Conversely, the *iaa18* mutant was found to have longer hypocotyls (Reed, 2001). The effect of these mutations on mature plant size and form (e.g. stem elongation and branching) remains unclear.

Aux/IAA genes also have a strong effect on apical dominance in inflorescence stems. For example, *iaa17/axr3-1* mutants have increased apical dominance (Leyser *et al.*, 1996) with fewer side branches while *iaa28-1* (Rogg *et al.*, 2001) has decreased apical dominance with more inflorescence branching. Lateral root branching is also affected in several mutants –



iaa3/shy2-2 (Tian & Reed, 1999; Tian *et al.*, 2002), *iaa14/slender (slr)-1* (Fukaki *et al.*, 2002) and *iaa28-1* (Rogg *et al.*, 2001) produce fewer root branches while *iaa17/axr3-1* (Leyser *et al.*, 1996; Rouse *et al.*, 1998) plants display more lateral roots than wild-type plants. Loss-of-function mutations in *Aux/IAA* genes seem to condition very subtle phenotypes likely because of redundancy and/or a feedback mechanism. However, antisense suppression of *IAA9* in tomato (*Lycopersicon esculentum*) produced numerous growth and form alterations (Wang *et al.*, 2005; Fig. 1b). Wild-type compound leaves were transformed into simple leaves, stem/hypocotyl elongation was enhanced, and apical dominance was reduced.

Auxin response factor (*ARF*) genes The founding member of the *ARF* gene family, *ARF1*, was discovered using the yeast one hybrid system because of its property to bind to the auxin response *cis*-element (*AuxRE*) found in the promoters of many auxin-regulated genes (Ulmasov *et al.*, 1997). ARFs can be activators or repressors of transcription, depending on the nature of a central protein domain (Ulmasov *et al.*, 1999; Tiwari *et al.*, 2003). They bind to DNA to regulate transcription as homo- or heterodimers with other ARFs or Aux/IAA proteins (Liscum & Reed, 2002). Loss-of-function mutations in a few *ARF*s have strong and specific phenotypes, including effects on stature, leaf morphology, and root architecture (Okushima *et al.*, 2005b). Three Arabidopsis null *ARF2* alleles, generated by T-DNA insertions and identified using a reverse genetics approach, produce plants that display longer, thicker inflorescence stems, and larger, darker green leaves compared with wild-type plants (Okushima *et al.*, 2005a). In addition to stem and leaf enlargement, *arf2* seeds were also larger than wild-type seeds. *ARF2* overexpression and RNAi suppression resulted in transgenic plants that phenocopied the *arf2* mutant, a result of cosuppression and RNAi downregulation, respectively.

Although many of the *ARF* single loss-of-function mutants do not show growth and developmental defects, presumably because of functional redundancy among the 23 members, some of the double mutations have a significant effect on stature and form (Okushima *et al.*, 2005b). For example, *arf19* is phenotypically indistinguishable from wild-type plants. *ARF7* is the putative paralog of *ARF19* and the double *arf7/arf19* mutant displays thin and short florescence stems, enhanced apical dominance, and reduced and delayed lateral root formation (Okushima *et al.*, 2005b). Overexpression of *AFR19* in transgenic plants produces a distinctive dwarf phenotype, decreased apical dominance, and narrow, elongated leaves (Okushima *et al.*, 2005b).

Ubiquitin-mediated regulatory degradation Regulated protein degradation plays an essential role in auxin signaling (Dharmasiri & Estelle, 2004; Leyser, 2002). The central role of this mechanism in auxin signaling is exemplified by the discovery that the auxin receptor is part of the ubiquitination pathway that leads to protein degradation (Dharmasiri *et al.*, 2005; Kepinski & Leyser, 2005). Several mutants affected in components of the pathway can display strong modifications in stature and form. For example, the *axr1-12* mutant of Arabidopsis harbors a loss-of-function mutation in a gene encoding the amino-terminal part of a ubiquitin-activating enzyme (Leyser *et al.*, 1993; Stirnberg *et al.*, 1999). Recently, two ubiquitin C-terminal hydrolases (UCHs) (i.e. UCH1 and UCH2) that are involved in de-ubiquitination and reversing the effect of ubiquitin conjugation were also found to be involved in auxin signaling through increasing or decreasing AUX/IAA protein stability, respectively, in overexpressing and loss-of-function mutants (Yang *et al.*, 2007). Overexpressing plants increased, while double mutants suppressed, the outgrowth of cauline lateral branches.

2. Gibberellin – the 'Green Revolution' hormone

Gibberellins (GAs) are a complex family of tetracyclic diterpenoid growth regulators that play a critical role in many plant growth and developmental processes (reviewed in Hooley, 1994; Davies, 1995). Advances in molecular genetics

Fig. 1 (a) Arabidopsis *Arabidopsis vacuolar pyrophosphatase1* (*AVP1*) overexpression phenotypes, with the wild type (WT) shown on the left and two independent *AVP1*-overexpressing lines on the right (reproduced from Li *et al.,* 2005b with permission from The American Association for Advancement in Science (AAAS)). Bar, 1 cm. (b) Tomato (*Lycopersicon esculentum*) phenotypes produced by down-regulation of the gene encoding an AUX/IAA transcription factor *IAA9*; the wild type is shown on the left, an antisense (AS) mutant in the middle, and a monogenic spontaneous *entire* putative *iaa9* mutant on the right (AC, Ailsa Craig; reproduced with permission from Wang *et al.*, 2005, ©American Society of Plant Biologists). Bar, 100 mm. (c) Dwarf field phenotype (foreground) of an activation tagged mutant in poplar resulting from hyperexpression of the catabolic gibberellin oxidase gene *PtaGA2ox1* (Busov *et al.,* 2003). Poplars (*Populus tremula* × *alba*) showing wild-type growth after two growing seasons are in the background (approx. 5 m in height). (d) Arabidopsis *ARGOS* (auxin-regulated gene controlling organ size) mutant phenotypes (reproduced with permission from Hu *et al.,* 2003, ©American Society of Plant Biologists). Antisense knock-down (left), vector control (middle), and 35S overexpression (right) mutants are shown. Bar, 5 mm. (e) Upper row, branching morphologies of maize (*Zea mays*) (left) vs its ancestor teosinte (right); lower row, segregation of form among recombinant inbred progeny derived from maize–teosinte hybridization that are homozygous for maize (left) or teosinte (right) chromosomal segments containing the major quantitative trait loci (QTL) for branching with the *TEOSINTE BRANCHED1* (*TB1*) gene. (Provided by, and used with permission from, J. Doebley.) (f) Arabidopsis *branched1* (*brc1*) and *branched2* (*brc2*) mutant phenotypes (reproduced with permission from Aguilar-Martinez *et al.,* 2007, ©American Society of Plant Biologists). (g) Arabidopsis *jaw* miRNA mutant phenotypes; the wild type is shown on the left, and the mutant on the right (reproduced with permission from Macmillan Publishers Ltd, from Palatnik *et al.,* 2003). (h) Tomato mutants for the *SELF-PRUNING* gene; the wild-type indeterminate form is shown on the left, and the homozygous determinate mutant on the right (AS, axillary shoot; TI, terminal inflorescence; from Pnueli *et al.,* 1998; reproduced with permission of the Company of Biologists).

have allowed identification of many of the genes involved in the metabolism and signaling pathway, and dissection of their role in regulation of plant stature and form (reviewed in Hedden & Phillips, 2000a; Sun, 2000; Olszewski *et al.*, 2002). The results of these studies were eloquently summarized in a recent review article as 'a tale of the tall and the short' (Thomas & Sun, 2004). Typically, mutants with a deficiency in GA concentrations or response are dwarf or semi-dwarf in stature, while elevated GA concentrations or increased signaling result in taller plants. GA metabolic and response genes have provided the basis of the 'Green Revolution' varieties of rice and wheat (David & Otsuka, 1994) and have been a logical focus for improving crop performance via both conventional breeding and genetic engineering (Sakamoto *et al.*, 2003). The *Reduced height1* (*Rht1*) allele in wheat is a dominant gain-of-function mutation in the coding sequence of a DELLA protein (discussed below in 'Negatively acting components'; Peng *et al.*, 1999), while the *semidwarf1* (*sd1*) 'Green Revolution' allele in rice is a recessive loss-of-function mutation in one of the major GA biosynthetic genes – GA20-oxidase (*GA20ox*) (Monna *et al.*, 2002; Sasaki *et al.*, 2002; Spielmeyer *et al.*, 2002).

Several properties of GA metabolic and response genes make them particularly attractive targets for manipulation. First, many of the genes act in a dose-dependent manner, allowing generation of a gradient of phenotypic responses (Cowling *et al.*, 1998). Secondly, similar phenotypic effects can be readily achieved in heterologous species (Hynes *et al.*, 2003; Busov *et al.*, 2006). Finally, in contrast to most other plant hormone modifications, the pleiotropic effects are usually positive with respect to crop performance – including increased nitrogen assimilation (Nagel & Lambers, 2002), photosynthesis (Biemelt *et al.*, 2004), and lateral root production (Busov *et al.*, 2006).

Metabolic genes GAs are synthesized in three successive steps localized in separate intracellular compartments, with the first stage in chloroplasts, the second in the endoplasmic reticulum, and the third in the cytoplasm (Hedden & Phillips, 2000a). The flux of bioactive GAs is controlled by the enzymes in the third compartment, such as GA20ox, GA3-oxidase (GA3ox), and GA2-oxidase (GA2ox) (Hedden & Phillips, 2000b). GA20ox and GA3ox are biosynthethic enzymes that catalyze the last two steps in the biosynthetic pathway. Until recently, GA2ox was the only known GA-inactivating enzyme (Olszewski *et al.*, 2002); a new deactivation reaction that is catalyzed by a P450 enzyme was recently described in rice (Zhu *et al.*, 2006). Each of these enzymes is encoded by a small family or subfamily of genes. Loss-of-function mutations in the *GA20ox* and *GA3ox* genes or overexpression of the *GA2ox* genes has a dwarfing effect and has been observed in numerous plant species, including Arabidopsis (Sun & Kamiya, 1994; Helliwell *et al.*, 1998; Yamaguchi *et al.*, 1998), rice (Sakamoto *et al.*, 2001), potato (*Solanum tuberosum*; Carrera *et al.*, 2000), and poplar (*Populus tremula* × *alba*; Busov *et al.*, 2003; Fig. 1c) (reviewed in Hedden & Phillips, 2000b). By contrast, GA-overproducing mutants, with hyperactivated GA biosynthetic activity, or reduced activity of the catabolic genes, often show extreme shoot elongation (Martin *et al.*, 1999; Carrera *et al.*, 2000).

Signal transduction mutants Genes involved in the GA signal transduction pathway have been identified through GA response mutants. These mutants are either GA-insensitive dwarfs or constitutive GA response mutants (reviewed in Sun, 2000). GA-insensitive mutants show symptoms of GA deficiency, but unlike GA metabolic mutants cannot be rescued by GA treatment. The signaling components identified through such mutations can be broadly classified into positively and negatively acting groups.

Positively acting components. These represent a diverse group of genes encoding heteromeric G proteins, transcription regulators, chromatin-remodeling factors, and enzymes. Loss-of-function mutations in some of these genes cause distinct dwarf or semi-dwarf phenotypes. *DWARF1* (*D1*) in rice is the only gene that encodes an α subunit of the heteromeric G protein (Ashikari *et al.*, 1999). Knockouts of the gene cause reduced stature and dark-green leaves, similar to GA-deficient rice plants. The first leaf in *d1* plants is GA insensitive but the second leaf shows a normal GA response. Knockout mutations in the Arabidopsis ortholog, although this is also a single-copy gene, do not result in the dwarf phenotypes observed in rice (Ullah *et al.*, 2001). The normal GA sensitivity of the second leaf in rice and the lack of dwarf phenotype in Arabidopsis suggest that D1 may not be directly involved in GA signaling, and that its importance in GA signal transduction varies among species.

PHOTOPERIOD RESPONSIVE1 (*PHOR1*) was identified in potato in a screen for mRNAs that accumulate during short day (SD) inductive treatment (Amador *et al.*, 2001). Antisense knockouts of the gene cause a semi-dwarf phenotype similar to that of GA metabolic mutants, and overexpression results in enhanced growth. Sequence predictions and PHOR1::GFP fusion experiments suggest that PHOR1 is a transcription factor that is regulated through modification of a Cys-Pro-Ile (CPI) domain, resulting in differential accumulation in the nucleus under GA signaling, and sequestration in the cytosol in the absence of GA signaling.

SLEEPY is a gene that was initially identified as a suppressor of the Arabidopsis abscisic acid insensitive mutant *abi1-1*, and its loss-of-function causes dwarf phenotypes and dark-green foliage typical of mutants associated with GA signaling or metabolism (Steber *et al.*, 1998). The corresponding gene was subsequently cloned and found to encode an F-box subunit of an Supressor of kinetochore protein1/Cullin/F-box protein complex (SCF) E3 ubiquitin ligase that participates in ubiquitination of proteins targeted for degradation, with the putative targets being DELLA proteins.

Negatively acting components. These include_mutations in *GA INSENSITIVE* (*GAI*), *REPRESSOR OF GA1* (*RGA*), and *RGA-LIKE1* (*RGL1*). These genes have been identified in many plant species, including Arabidopsis (Peng *et al.*, 1997; Silverstone *et al.*, 1997; Lee *et al.*, 2002; Wen & Chang, 2002), rice (Ikeda *et al.*, 2001), wheat (Peng *et al.*, 1999), maize (Peng *et al.*, 1999) and grapevine (*Vitis vinfera*; Boss & Thomas, 2002). Gain-of-function mutations in these genes cause a semi-dominant dwarf phenotype, while loss-of-function mutations are recessive and result in increased growth. Mutant analyses of these proteins suggest that they are negative regulators of the GA signal transduction pathway.

GAI, RGA and RGL belong to the larger GAI, RGA and SCARECROW (GRAS) family of transcription factors and are also known as DELLA proteins because of a conserved N-terminus DELLA domain that is absent in the other family members (Pysh *et al.*, 1999). Complete deletion or nonsynonymous substitutions in this domain produce strong gain-of-function, dominant mutations that result in constitutive inhibition of one or several GA responses (Peng *et al.*, 1997). Such mutations result in dwarf or semi-dwarf plants and similar effects of transgenic expression of the mutant forms can be observed in heterologous species (Fu *et al.*, 2001; Busov *et al.*, 2006). As discussed above, natural mutations identified in rice and wheat through traditional breeding became the basis for the development of the 'Green Revolution' varieties (Silverstone & Sun, 2000).

SPINDLY (SPY) is also believed to be a negative regulator of the GA response in plants (Jacobsen *et al.*, 1996; Thornton *et al.*, 1999a). Constitutive overexpression of the Arabidopsis *SPY* gene in Arabidopsis (Swain *et al.*, 2001) and petunia (*Petunia hybrida*; Izhaki *et al.*, 2002) causes dwarfing. SPY shows protein sequence similarity to UDP-GlcNAc protein transferases (OGTs) in animals (Thornton *et al.*, 1999b), and has been demonstrated to possess OGT activity (Thornton *et al.*, 1999a). OGT protein modification regulates protein activity, and the extent of this modification depends on metabolic hormonal and developmental signals (Corner & Hart, 2000). *SHORT INTERNODES* (*SHI*) is part of a nine-member gene family that have RING finger-class zinc finger motifs, which have been suggested to play roles in protein–protein interactions during proteolysis or transcription activation (Fridborg *et al.*, 1999). Overexpression of the gene results in decreased shoot elongation, suggesting that *SHI* is also a negative regulator of GA responses (Fridborg *et al.*, 2001).

3. Brassinosteroids

Brassinosteroids (BRs) are a class of more than 40 sterol derivatives in plants that have profound effects on plant size and architecture. Biosynthesis and signal transduction have been subjects of intense genetic dissection (reviewed in Fujioka & Yokota, 2003; Vert *et al.*, 2005; Haubrick & Assmann, 2006). Several mutations found recently hold promise for modification of stature and form relevant to crop improvement (Bishop, 2003), and are summarized below.

The rate-limiting biosynthetic and catabolic steps in BR metabolism have been identified in BR-deficient mutants. The classic BR-deficient phenotype is characterized by short robust stems, and small, round, dark-green leaves. The C6- and C22α-oxidation steps are rate limiting in synthesis of brassinolide – the most bioactive BR found to date (Choe *et al.*, 2001). The tomato *DWARF* gene *D* was isolated via transposon tagging and found to show homology to two P450s (CYP90A and CYP90B) and was classified as CYP85 (Bishop *et al.*, 1999). Mutant plants showed classic BR phenotypes but, unlike in Arabidopsis, did not display reduced apical dominance. Overexpression of the gene under the 35S promoter fully complemented the *dwarf* allele, and the lines were larger than wild type; however, a limited number of lines were screened, precluding general conclusions on its growth-enhancing effects. More conclusive results with respect to the growth-promoting effects of these genes were obtained in a study of the *DWARF4* (*DWF4*) gene in Arabidopsis (Choe *et al.*, 2001). *DWARF4* was found to encode a P450 enzyme with highest homology to the Arabidopsis CONSTITUTIVE PHOTOMORPHOGENESIS DWARFISM (CPD) protein (CYP90A1). Loss-of-function produced a dwarf phenotype, while overexpression caused strong growth-promoting effects in both Arabidopsis and tobacco (*Nicotiana tabacum*) that was similar to that of exogenously applied bioactive BR. The height of transgenic Arabidopsis plants was 40% greater than that of wild-type plants and resulted primarily from continued growth beyond 35 d after germination, when wild-type plants had ceased elongation. Height was similarly but more modestly (14%) increased in tobacco. In both Arabidopsis and tobacco, *DWF4* increased petiole and leaf blade length and increased lateral branching.

In cereals, BR deficiency is associated with an increase in leaf erectness, which is an important crop trait because it increases photosynthesis in lower leaves, yet allows normal growth under dense planting conditions on farms (Feldmann, 2006). In contrast to Arabidopsis, *DWARF4* in rice is encoded by two genes, *OsDWARF4L1* and *OsDWARF4* (Sakamoto *et al.*, 2006). The two genes encode enzymes of redundant biochemical functions but of very different developmental roles, likely because of their different expression patterns. For example, *OsDWARF4L1* loss-of-function results in semi-dwarf phenotypes with small seeds, while knockout mutations in *OsDWARF4* cause more modest dwarfing, do not affect seed size, and increase leaf erectness. A small field trial experiment with plants carrying the *osdwarf4-1* mutant allele with two planting densities and three levels of nitrogen revealed that, under highest density and nitrogen, the *osdwarf4-1* plants produced 40% more biomass. The *osdwarf4-1* plants displayed increased grain yields (17–20%)

compared with wild-type plants at all nitrogen levels under a dense planting environment. Differences in both biomass and grain yield were less dramatic under normal planting density.

4. A novel hormonal pathway controls branching

Plant form is largely determined by the activity of axillary meristems, whose growth is regulated by auxin and CK. In many plant species, shoot apices grow predominantly and repress axillary bud growth, a process termed apical dominance (e.g. Cline, 2000). Shoot tips produce the majority of auxin, and thus removal (decapitation) typically induces outgrowth of axillary buds; application of auxin to the cut tip prevents outgrowth. Moreover, application of auxin transport inhibitors to the stems of intact plants can reduce apical dominance, further supporting the hypothesis that apically derived auxin is transported basipetally and inhibits outgrowth of axillary buds. By contrast, application of CK to axillary buds or to roots often promotes outgrowth. In addition to modification of the form of annual plants, genes that affect auxin signals are likely to affect apical dominance in trees, which is an important determinant of wood quality, fruit yield, and biomass production (e.g. Bradshaw & Strauss 2001).

Studies in Arabidopsis and other annual plants have identified a novel hormonal pathway regulating the outgrowth of axillary meristems (reviewed in McSteen & Leyser 2005; Bennett & Leyser, 2006; Dun *et al.*, 2006). Reciprocal grafting studies between mutant and wild-type pea (*Pisum sativum*) showed that regulation of bud outgrowth involves long-distance signaling that does not involve auxin and CK (Dun *et al.*, 2006). In Arabidopsis, four genes, *MORE AXILLARY BRANCHING* (*MAX)1–4*, have been identified that are involved in this signaling pathway; they act to repress lateral outgrowth. *MAX3* and *MAX4* are required for the production of a yet unidentified graft transmissible branching signal, and belong to the carotenoid cleavage dioxygenase (CCD) family, suggesting that this signal might be a carotenoid derivative (Sorefan *et al.*, 2003; Booker *et al.*, 2004). *MAX1* acts downstream of *MAX3/4* in the synthesis of the branching signal and encodes a member of the cytochrome P450 family (Booker *et al.*, 2005). *MAX2* encodes an F-box protein, which is typically involved in ubiquitin-mediated protein degradation – a common strategy employed in plants for signal perception and transduction (Stirnberg *et al.*, 2002). MAX2 interacts with the core components of SCF-type E3 ubiquitin ligases and acts locally at the node (Stirnberg *et al.*, 2002).

Mutant and reciprocal grafting analyses have also revealed a similar inhibitory pathway in pea and petunia, controlled respectively by the *RAMOSUS* (*RMS*) and *DECREASED APICAL DOMINANCE* (*DAD*) genes (e.g. Dun *et al.* 2006; Simons *et al.*, 2007). *RMS5*, *RMS1* and *RMS4* have been cloned and are orthologs of *MAX3*, *MAX4* and *MAX2*, while *DAD1* is orthologous to *MAX4*/*RMS1* (Sorefan *et al.*, 2003; Snowden *et al.*, 2005; Johnson *et al.*, 2006). Recently, rice orthologs of *MAX2*, *MAX3* and *MAX4* have been shown to repress tiller bud outgrowth (Ishikawa *et al.*, 2005; Zou *et al.*, 2006; Arite *et al.*, 2007). Although this pathway is conserved among diverse angiosperms, differences are also apparent. For example, *RMS1* expression is altered in different *rms* mutant backgrounds and expression in the stem is affected by auxin concentrations (Foo *et al.*, 2005), but such regulation was not observed for the orthologous *MAX4* (Sorefan *et al.* 2003; Bainbridge *et al.*, 2005). Whether these and other differences reflect major differences in this pathway between species, variation in the importance of various pathway components or differences in experimental techniques remains to be determined (Dun *et al.*, 2006; Ongaro & Leyser, 2007). Studies by Bennett *et al.* (2006) suggested that the MAX pathway acts by controlling auxin transport capacity in the stem. With the exception of *rms2*, pea *rms* mutants as well as Arabidopsis *max* mutants show reduced xylem sap CK but have near wild-type concentrations of shoot CK (Foo *et al.*, 2007). Detailed studies in pea have provided strong evidence for a basipetally moving feedback signal involving *RMS2* that reduces xylem CK and promotes the expression of *RMS1* and *RMS5*. Although the identity of this feedback signal and many other details are still unknown, the *MAX/RMS* pathway clearly involves cross-talk with auxin and cytokinin.

III. Transcription factors and other regulatory genes

1. The *AINTEGUMENTA* (*ANT*) and ARGOS pathway

One of the main controllers of plant organ size is *AINTEGUMENTA* (*ANT*). Originally discovered as a result of its effect on flower development (Elliott *et al.*, 1996; Klucher *et al.*, 1996), *ANT* overexpression has been shown to dramatically increase both leaf and floral size in Arabidopsis (Krizek, 1999; Mizukami & Fischer, 2000). The increase in flower size in *35S::ANT* expressing plants was manifested in both the Columbia (Col) and Landsberg erecta (L-er) ecotypes but the leaf size promoting effect was not described in the L-er ecotype. ANT is an APETALA2 (AP2)-domain transcription factor that negatively regulates *AGAMOUS* (*AG*) expression during flower development. There are seven *ANT*-like (*AIL*) genes in Arabidopsis, some of which have similar growth and size promoting effects (Nole-Wilson *et al.*, 2005). The size increase is associated with increased cell proliferation rather than cell size, suggesting that the gene prolongs the meristematic capacity of cells during organ growth and differentiation.

Recently, ANT was found to be part of an auxin-regulated signaling cascade. A central gene in this cascade, called *ARGOS* (auxin-regulated gene controlling organ size), was

discovered during microarray analysis of auxin response (Hu *et al.*, 2003). *ARGOS* has a similar growth-promoting effect as *ANT* when overexpressed in transgenic plants (Fig. 1d). ARGOS appears to be upstream of ANT in the signaling cascade, as loss of ANT function blocks the growth-promoting effect of ARGOS.

2. TCP transcription factors

The TCP-domain proteins are plant-specific transcription factors that regulate shape and form characteristics of plants (Cubas *et al.*, 1999). The TCP domain is a basic-helix-loop-helix secondary protein structure involved in DNA binding and protein dimerization, named after the three founding members of the family – *TEOSINTE BRANCHED1* (*TB1*) from maize, *CYCCLOIDEA* (*CYC*) from *Antirrhinum*, and *PROLIFERATION CELL FACTOR1* (*PCF1*) from rice. In Arabidopsis the family consists of 24 members that play important roles in branching, floral symmetry, and leaf curvature by synchronization of cell division and growth, likely by binding to promoters and transcriptionally regulating genes involved in the cell cycle and ribosomal machinery (Li *et al.*, 2005a).

The importance of this gene family in the evolution of modern crop plants is exemplified by the *TB1* gene from maize, which was the first domestication gene identified in any species (Fig. 1e). Its mutant form made a major contribution to development of modern maize from its wild teosinte ancestor (Doebley *et al.*, 1995, 1997). Overexpression of the *TB1* gene suppresses lateral branching and contributes to a more compact plant form suitable to cultivation under a high-density crop environment. *TB1* orthologs with similar phenotypic effects have been found in rice (*OsTB1*) and Arabidopsis (*BRANCHED1* and *BRANCHED2*) (Takeda *et al.*, 2003; Aguilar-Martinez *et al.*, 2007; Fig. 1f).

In *Antirrhinum*, the *CINCINNATA* gene encodes a TCP-domain transcription factor that controls leaf curvature and therefore surface features (Crawford *et al.*, 2004). Plants bearing homozygous *cin* alleles show excessive curvature, particularly in the marginal regions, resulting from excessive uncontrolled cell growth. Developing leaves in *cin* mutants suffer from a delay in cell division arrest, resulting in an excess of cells, causing curvatures in the leaf surface. A similar effect is observed in Arabidopsis. Activation tagging of microRNA159/319 in the *jaw* mutant is a near phenocopy of the *cin* mutant in snapdragon (*Antirrhinum majus*; Palatnik *et al.*, 2003; Fig. 1g). The phenotypic effect is a result of miR159/319-mediated cleavage of *TCP4* mRNA. A *TCP* gene also controls development of compound leaves in tomato. Gain-of-function mutations in the *LANCEOLATA* (*LA*) gene render *LA* mRNA resistant to degradation by miR159/319 (Ori *et al.*, 2007). The elevated *LA* transcript abundance leads to precocious differentiation of the leaf margin and precludes compound leaf development.

3. Growth-regulating factor (*GRF*) and ANGUSTIFOLIA act together in control of leaf size and shape

Growth-regulating factor (*GRF*) genes represent a small transcription factor gene family in plants that control leaf size and lateral:longitudinal dimensions via control of cell proliferation. The founding member of the gene family, *OsGRF1*, was found in rice; it is induced by GA and is highly expressed in rapidly elongating stems (van der Knaap *et al.*, 2000). In Arabidopsis, three members of the family (*GRF1*, *GRF3*, and *GRF5*) were found to have strong growth-promoting effects on both leaves and cotyledons (Kim *et al.*, 2003). Loss-of-function studies indicate functional redundancy for some family members but not for others. For example, triple insertional knockout mutants of *GRF1–3* have smaller leaves and cotyledons, while single mutants have no change in phenotype. By contrast, a single knockout of *GRF5* exhibits a narrow leaf phenotype that is associated with a decrease in cell numbers (Horiguchi *et al.*, 2005).

Yeast two hybrid system studies have demonstrated that GRF proteins interact with ANGUSTIFOLIA3 (AN3) via their N-terminal domains. *AN3* encodes a protein similar to a human transcriptional coactivator, synovial sarcoma translocation protein (SYT) (Kim *et al.*, 2002; Kim & Kende, 2004; Horiguchi *et al.*, 2005). Modification of *AN3* expression seems to have phenotypic effects similar to that for *GRF*, suggesting that the two proteins may act together to control the development of leaf size and shape. Overexpression of *AN3* in transgenic plants under a strong 35S promoter induces large leaves, while its knockout results in small, narrow leaves.

4. GRAS and MYB proteins control branching

Molecular and genetic analyses in annuals have shown that control of branching involves at least two steps, the formation of the axillary meristem and outgrowth of the axillary bud (Schmitz & Theres, 2005). The tomato genes *BLIND* (*Bl*), which encodes a MYB-domain transcription factor, and *LATERAL SUPPRESSOR* (*Ls*), which encodes a GRAS-domain transcription factor, control the initiation of lateral meristems (Schumacher *et al.*, 1999; Schmitz *et al.*, 2002). Studies of *Ls* homologs from rice (*MONOCULM1* (*MOC1*); Li *et al.*, 2003) and Arabidopsis (*LATERAL SUPPRESSOR* (*LAS*); Greb *et al.*, 2003) as well as *Bl* homologs from Arabidopsis (*REGULATOR OF AXILLARY MERISTEMS* (*RAX*)*1/2/3*; Keller *et al.*, 2006; Muller *et al.*, 2006) support the idea that these are key regulators of axillary meristems conserved over large evolutionary distances. *Ls/LAS* and *Bl/RAX1* appear to act in separate pathways and have partially redundant functions in axillary meristem initiation and maintenance. There are, however, also important differences among the homologs. For example, *ls* mutants lack branches during the vegetative phase, but produce branches after the plant has transitioned to flowering.

By contrast, *moc1* mutants show large reductions in branching in both vegetative and inflorescence shoots. *Bl* regulates axillary meristem initiation during both vegetative and reproductive development, whereas *RAX1/2/3* regulates branching along overlapping zones of the shoot, with *RAX1* acting early in vegetative development and *RAX2/3* primarily acting later during inflorescence development. *RAX1* also appears to affect the timing of the floral transition by modulating GA concentrations in the shoot apex. These results demonstrate the effects that gene duplication or loss and subsequent subfunctionalization can have in altering regulatory networks, and show that gene regulation within axillary meristems can change after developmental phase transitions.

5. *TERMINAL FLOWER1* (*TFL1*) and shoot architecture

The Arabidopsis gene *TFL1* controls shoot meristem identity throughout the plant life cycle, and encodes a putative signal transduction protein with homology to mammalian phosphatidylethanolamine-binding proteins (Bradley *et al.*, 1997). Overexpression of *TFL1* results in increased vegetative growth, a larger and highly branched inflorescence, and delayed flower formation (Ratcliffe *et al.*, 1999). By contrast, *tfl1* mutants have a short vegetative phase during which they produce few leaves and branches, and the normally indeterminate inflorescence meristem forms a terminal flower (Shannon & Meeks-Wagner, 1991). *TFL1* opposes the activity of the floral meristem identitiy gene *LEAFY* (*LFY*) (Liljegren *et al.*, 1999; Ratcliffe *et al.*, 1999). The central role of these two genes in determining shoot meristem fate provided compelling support for a model explaining how developmental genetic mechanisms constrain the evolution of inflorescence architecture (Prusinkiewicz *et al.*, 2007). For example, the relative rarity of mutations that cause changes in the regulatory interactions of *LFY* and *TFL1* compared with changes that result in reduction in gene function may account for the rarity of genera that include species with both racemes and cymes.

Natural and induced mutations in the pea *TFL1* homolog *LATE FLOWERING* (*LF*) have been important for pea domestication (Weller *et al.*, 2007). Three of four *LF* alleles do not show variation in encoded protein sequence but rather vary in expression of *LF* (Foucher *et al.*, 2003). For example, the dominant *Lf-d* allele results in an increased amount of *LF* transcript, which delays flowering and gives rise to plants that have a higher number of lateral branches. A major domestication trait in tomato was produced by a recessive mutation in the tomato *TFL1* homolog *SELF-PRUNING* (*SP*) (Pnueli *et al.*, 1998; Fig. 1h). The *sp* allele causes an accelerated termination of sympodial units into inflorescences, resulting in a bushy, compact form and nearly homogeneous sized fruits. By increasing the uniformity in time of ripening and inducing a 'determinate' growth habit, the *sp* trait allowed the development of modern mechanical harvesting technology.

IV. Cell cycle genes: cause or consequence of growth?

The role of the cell cycle in plant growth and development has been a subject of considerable debate (Doonan, 2000; Mizukami & Fischer, 2000; Inze & Veylder, 2006). The cell cycle is obviously necessary for generating the cells that build organs and organisms; bigger plants and organs are generally built of more cells (Basile & Basile, 1993; Meyerowitz, 1997). However, is the cell cycle the driver of growth or is merely subordinate machinery filling predetermined developmental space? Is direct modulation of the cell cycle machinery useful for manipulating plant growth and development?

Early experiments showed that arresting cell division by gamma irradiation of wheat seedlings had little effect on final growth and development (Haber, 1962). The lower cell numbers were largely compensated by increases in cell size. Results from recent experiments with transgenic plants that had altered expression of various cell cycle regulatory molecules in general confirm the early findings, yet the results are highly variable (Beemster *et al.*, 2005; Inze & Veylder, 2006). The focus of manipulations has been the main checkpoints that determine the speed and synchronization of cell division (e.g. G1/S and G2/M transitions). The molecular controls determining progression through these transitions are represented by heteromeric cyclin-dependent kinases (CDKs) with their regulatory subunits: the activating unit is known as cyclin, and the inhibitory unit as cyclin-dependent kinase inhibitor (CKI/KRP) (reviewed in Dewitte & Murray, 2003; Verkest *et al.*, 2005; Inze & Veylder, 2006).

1. Cell proliferation

Results from experiments that directly slowed the rate of cell cycling have produced variable results. In many cases, effective decreases in cell proliferation resulted in fewer cells, but compensatory increases in cell size reduced most of the impact on final size and form (Doonan, 2000; Inze & Veylder, 2006). For example, expression of a dominant negative form of CDKA;1 in transgenic tobacco resulted in a block of G1/S transition and decreased cell proliferation; however, it was compensated by larger cell sizes, resulting in normal growth and development (Hemerly *et al.*, 1995). By contrast, overexpression of any of the *CKI/KRP* genes in Arabidopsis resulted in cell cycle retardation and fewer cells and, despite partial compensation through increases in cell size, transgenic plants were severely dwarfed, with most organs reduced in size. There were also modifications in plant morphology, such as in the shape and serration of leaves and petals (Wang *et al.*, 2000; de Veylder *et al.*, 2001). Transgenic plants with increased cell cycling via induction of faster G1/S transitions have increased growth rates in roots and/or shoots, but with no effects on overall plant stature. For example, overexpression of *CYCD2;1* in transgenic tobacco increased shoot growth and

accelerated development (Cockcroft *et al.*, 2000). Overexpression of B-type cyclins (*CYCB1;1* and *CYCB2;2*) increased the rate of root growth (Doerner *et al.*, 1996; Lee *et al.*, 2003).

Although such modifications do not result in changes of final size and form, accelerated growth rates can provide benefits to crops grown under short growing seasons.

2. Incomplete cell cycles: endoreduplication and growth

Normally, the cell cycle is characterized by a round of DNA duplication followed by mitosis and cytokinesis (Dewitte & Murray, 2003; Inze & Veylder, 2006). Many plant cells, and most prominently the endosperm and trichomes, undergo rounds of DNA replications without cytokinesis that are known as endoreduplication (Kondorosi *et al.*, 2000). In plants, increases in nuclear size caused by polyploidization are positively correlated with cell size, and consequently with organ and organism size (Kondorosi *et al.*, 2000). Therefore, genes that regulate endoreduplication have been viewed as potential targets for modifying cell size and consequently plant stature. This notion has also been supported by observations of plant species that naturally endoredulicate and also have accelerated development and stable yields (Barow & Meister, 2003; Jovtchev *et al.*, 2006).

However, similar to the experience with cell cycle gene manipulation discussed above, it also does not appear that alterations in the expression of the regulatory genes that control endoreduplication give rise to predictable and useful improvements in plant form and size. In many of these manipulations increased nuclear DNA content was observed, but this was likely a result of compensatory mechanisms; the final stature and form were not or only slightly changed (reviewed in Inze & Veylder, 2006). For example, KRP2, an inhibitor of CDKA1;1, controls the onset of the endoreduplication cycle in Arabidopsis (Verkest *et al.*, 2005). Transgenic plants overexpressing *KRP2* under the 35S promoter showed a dose-dependent response, with the highly expressing lines having no effect on the level of endoreduplication and ploidy, and the lines with low transgene expression showed higher levels of endoreduplication and ploidy in leaves. The overall size and form of transgenic plants were not changed. Expression of *KRP2* under the *SHOOT MERISTEMLESS (STM)* gene promoter, which drives expression in mitotically active cells, increased endoreduplication and ploidy levels in leaf cells. The total size of transgenic plants was slightly decreased because of mitosis inhibition, but the resulting decrease in cell number was almost fully compensated by increases in cell size.

Another well-documented regulator of endoreduplication is the cell cycle switch 52 (CCS52), isolated from *Medicago* and playing a role in cell enlargement during nodule formation (Cebolla *et al.*, 1999; Vinardell *et al.*, 2003). Likely because overexpression of *CCS52* causes embryonic lethality, no transgenic plants overexpressing the protein were recovered. Antisense-mediated down-regulation resulted in wild-type-like plants that were somewhat slender, a result of the formation of fewer side branches. A DP-E2F-like1 (DEL1) protein with an unknown molecular role seems to be negatively regulating the onset of endoreduplication (Vlieghe *et al.*, 2005). Although transgenic plants with down- or up-regulation of *DEL1* predictably changed the ploidy level, transgenics were phenotypically similar to wild-type plants.

V. Conclusions

1. Functional analysis of genes controlling size and form – discovery vs function

Although there are notable exceptions that include *AVP1* (Multani *et al.*, 2003), *PHOR1* (Amador *et al.*, 2001), and quantitative trait locus *FW2.2* (Frary *et al.*, 2000), the great bulk of identifications of genes, and analyses of their basic functions, have taken place in a few model plant species (Table 1). The species primarily used has been Arabidopsis, but rice and tomato have also played significant roles. Other organisms with good genomic resources and transgenic capabilities – such as poplar and the legumes – are capable of providing some additional discoveries (e.g. Zubko *et al.*, 2002; Busov *et al.*, 2003; Tuskan *et al.*, 2006; Ayliffe *et al.*, 2007). However, because of the tremendous variety of plant sizes, morphologies, and developmental pathways, it is also clear that even a basic picture of how gene function and regulation control this diversity cannot be obtained from models alone. With the major genes and pathways already identified, it is an opportune time to accelerate studies of evolutionary variation in plant form and function (Remington & Purugganan, 2002).

This would also seem to be an opportune time for functional genomics aimed at intensive dissection of the kinds of form/size regulation of most importance for crop systems and their products. Reverse and association genetic approaches in a set of 'second tier' models chosen to represent plant morphological, physiological, phylogenetic, and crop diversity would seem to be most profitable (Irish & Benfey, 2004; Pennisi, 2007). For example, genes affecting primary growth meristems have dominated studies of annual plant models. However, identification and functional characterization of genes affecting secondary (cambial) meristematic activity, which are responsible for production of xylem, cambium, phloem, and bark, have hardly begun. Woody tissues derived from the cambium support fundamental ecosystem services, and provide much of the commercial values of forests. Gene discovery affecting these traits will be more productive in second-tier model species such as poplar (Jansson & Douglas, 2007) – which has been productively used in both forward and reverse genetic studies, as well as in polymorphism-based chromosomal mapping and association genetic studies (Eriksson *et al.*, 2000; Busov *et al.*, 2003; Wu *et al.*, 2003). Species such as *Mimulus* spp. and *Aquilegia* spp. would be obvious choices for

Table 1 Genes with strong effects on plant stature or form

Gene	Biochemical function	Trait affected	Modification	Species	Reference
Hormonal control					
P-glycoprotein (*PGP*)	Auxin transport	Stature control in the first internode	Knockout	Maize (*Zea mays*)/ sorghum	Multani *et al.* (2003)
Arabidopsis vacuolar pyrophosphatase1 (*AVP1*)	Phosphatase	Increased leave size and increased number of rosette leaves	Overexpression	Arabidopsis	Li *et al.* (2005b)
Gibberellin20-oxidase (*GA20ox*)	GA biosynthesis	Increased stature and organ size Dwarfism when knocked out	Overexpression/ knockout	Many species	Hedden & Phillips (2000a)
Gibberellin3-oxidase (*GA3ox*)	GA biosynthesis	Increased stature and organ size Dwarfism when knocked out	Overexpression/ knockout	Many species	Hedden & Phillips (2000a)
Gibberellin2-oxidase (*GA2ox*)	GA catabolism	Dwarfism	Overexpression/ knockout	Many species	Hedden & Phillips (2000a)
DELLA proteins	Negative GA response	Dwarfism	Overexpression/ knockout	Many species	Olszewski *et al.* (2002)
PHOTOPERIOD RESPONSIVE1 (*PHOR1*)	Positive GA response	Increased stature and leaf size Dwarfism when knocked out	Overexpression/ knockout	Potato (*Solanum tuberosum*)	Amador *et al.* (2001)
SPINDLY (*SPY*)	Glycosylation enzyme	Dwarfism	Overexpression	Many species	Olszewski *et al.* (2002)
SHORT INTERNODE (*SHI*)	Negative GA response	Dwarfism	Overexpression	Arabidopsis/barley (*Hordeum vulgare*)	Fridborg *et al.* (2001)
DWARF	Brassinosteroid biosynthesis	Dwarfism and round leaves	Knockout	Tomato (*Lycopersicon esculentum*)	Bishop *et al.* (1999)
DWARF4	Brassinosteroid biosynthesis	Dwarfism. Increased stature, leaf size, and lateral branching when overexpressed	Knockout/ overexpression	Arabidopsis, tobacco (*Nicotiana tabacum*), rice (*Oryza sativa*)	Choe *et al.* (2001)
MORE AXILLARY BRANCHING1 (*MAX1*)	P450 enzyme	Negative regulation of branch outgrowth	Knockout	Arabidopisis	Booker *et al.* (2005)
MAX2	F-box protein	Negative regulation of branch outgrowth	Knockout	Arabidopisis, pea (*Pisum sativum*), petunia (*Petunia hybrida*), rice	Snowden *et al.* (2005); Johnson *et al.* (2006); Zou *et al.* (2006)
MAX3/4	Carotenoid cleavage dioxygenase	Negative regulation of branch outgrowth	Knockout	Arabidopisis, pea, petunia, rice	Snowden *et al.* (2005); Johnson *et al.* (2006); Zou *et al.* (2006)
Transcription factors and other signaling molecules					
AINTEGUMENTA (*ANT*)	AP2-domain transcription factor	Increased or decreased organ size when up- or down-regulated	Overexpression/ knockout	Many species	Krizek (1999); Mizukami *et al.* (2000)
AINTEGUMENTA-LIKE (*AIL*); seven members	AP2-domain transcription factor	Increased or decreased organ size when up- or down-regulated	Overexpression/ knockout	Arabidopsis	Nole-Wilson *et al.* (2005)
Auxin regulated gene controlling organ size (*ARGOS*)	Transcription factor	Increased organ size	Overexpression	Arabidopsis	Hu *et al.* (2003)
ARGOS-LIKE	Transcription factor	Increased organ size	Overexpression	Arabidopsis	Hu *et al.* (2006)
CINCINNATA (*CIN*)	TCP-domain transcription factor	Increased leaf curvature and surface	Knockout	Arabidopsis	Crawford *et al.* (2004)
TEOSINTE BRANCHED1 (*TB1*)	TCP-domain transcription factor	Increased apical dominance and decreased branch proliferation	Overexpression	Arabidopsis, maize, rice	Doebley *et al.* (1997); Takeda *et al.* (2003); Aguilar-Martinez *et al.* (2007)

Table 1 *continued*

Gene	Biochemical function	Trait affected	Modification	Species	Reference
LANCEOLATA (*LA*)	TCP-domain transcription factor	Transformation of compound to simple leaves	Gain-of-function degradation-resistant	Tomato	Ori *et al.* (2007)
Growth regulating factor (*GRF5*)	Transcription factor	Increased or decreased organ size when up- or down-regulated	Overexpression/ knockout	Arabidopsis	Horiguchi *et al.* (2005)
ANGUSTIFOLIA3 (*AN3*)	Transcription factor	Increased leaf size when up-regulated. Narrow leaves when down-regulated	Overexpression/ knockout	Arabidopsis	Horiguchi *et al.* (2005); Kim & Kende (2002, 2004)
BLIND (*Bl*)	Myb transcription factor	Reduced branching	Knockout	Arabidopsis, tomato	Schmitz *et al.* (2002); Muller *et al*. (2006); Keller *et al*. (2006)
LATERAL SUPPRESSOR (*Ls*)	GRAS transcription factor	Reduced branching	Knockout	Arabidopsis, tomato, rice	Schumacher *et al*. (1999); Li *et al*. (2003); Greb *et al*. (2003)
SELF-PRUNING (*SP*)	TFL1 ortholog	Determinate growth habit	Knockout	Tomato	Pnueli *et al.* (1998)
FW2.2	Signal transduction	Negative regulation of fruit size	Overexpression	Tomato	Frary *et al.* (2000)
JAWS	miR159/319	Increased leaf surface/ curvature	Overexpression	Arabidopsis	Palatnik *et al.* (2003)
Cell cycle genes					
CyclinD2 (*CYCD2*)	Regulation of G1/S transition	Increased growth rates but final size the same	Overexpression	Tobacco	Cockcroft *et al.* (2000)

GRAS, GAI, RGA and SCARECROW; TFL, TERMINAL FLOWER.

studies of evolution of floral morphology, and of rapid speciation and adaptation (Whittall & Hodges, 2007; Wu *et al.*, 2007). Other species will be of value for the study of size/form control in more specialized traits, such as for tuberization, fruit shape and size, and morphological changes associated with nitrogen fixation.

2. From mutant extremes to useful traits

The extreme mutant phenotypes that are generally produced via mutagenesis in model organisms are informative with respect to basic gene function, but are rarely useful in crop improvement (Morgante & Salamini, 2003). For example, the commercial 'Green Revolution' varieties of rice and wheat identified via conventional breeding are semi-dwarf, and have normal grain yields. These alleles are conditioned by mild forms of native alleles of gibberellin biosynthetic and signaling genes (Peng *et al.*, 1999; Spielmeyer *et al.*, 2002). The modest phenotypes of these varieties are in sharp contrast to complete loss-of-function or constitutive gain-of-function mutants used in most transgenic experiments – nearly all of which produce extreme phenotypes and unacceptable levels of pleiotropy (Olszewski *et al.*, 2002; Hedden & Phillips, 2000b).

Apart from potted ornamental plants and trees, alleles that impart more moderate types of variation will be necessary for generation of varieties for commercial deployment. How can such alleles be obtained? Conventional breeding is certainly a viable option, with the main limits being the costs of large screens, especially for difficult to assess traits such as those related to wood or root characteristics, or where stand-level performance must be assessed for thousands of accessions. In addition, to the extent that such alleles tend to be recessive or partially recessive and have a strong deleterious effect in wild populations, they may be difficult to identify in outcrossing species such as trees and many grasses. For example, dominant or semi-dominant alleles for dwarfism that do reduce plant vigor unacceptably may be difficult to find in wild populations because they will have been under strong negative selection (Jennings & Aquino, 1968; Nagano *et al.*, 2005).

DNA markers can be used to search for polymorphisms in native or breeding populations (Alonso-Blanco & Koorneef, 2000; Koornneef *et al.*, 2004). Although identification of DNA polymorphism in genes of interest has become increasingly rapid and affordable, substantial practical challenges remain (Morgante & Salamini, 2003). For example, it is challenging and costly to assemble large, replicated populations where many genes segregate in a manner that enables reliable statistical detection (Nadeau & Frankel, 2000; Morgante & Salamini, 2003). Full genome scans are still not feasible in most species, and would require a very large number of markers because of the low linkage disequilibrium in outcrossing species (Morgante & Salamini, 2003; Neale & Savolainen,

2004). However, as genotyping costs continue to diminish, statistical tools increase in power, and large specialized mapping populations are created, association methods may begin to provide the power needed for reliable gene identification.

Targeted induced local lesions in genomes (TILLING) holds promise for overcoming some of the problems associated with limited natural variation and allele strength. This technology employs the property of a Celery endonuclease1 (CEL1) enzyme from celery (*Apium graveolens*) to recognize and cleave mismatches between different allelic copies of a gene (McCallum *et al.*, 2000). Induced point mutations in genes of interest produced using chemical or physical mutagens can thus be easily screened, and subsequently tested *in planta* if homozygous genotypes can be produced via inbreeding (most lesions cause recessive, loss-of-function alleles). For example, wheat granule bound starch synthase 1 (known as the *waxy* locus) produces waxy starch, lacking in amylase, that has unique physicochemical properties. Despite the commercial interest in deploying waxy varieties in wheat, a lack of variation at the *waxy* loci of interest prevented deployment by conventional breeding methods. Using TILLING, 250 artificially induced allelic variants were evaluated and subsequently used to generate the desired phenotype (Slade *et al.*, 2005).

Transgenic approaches can also be used to provide more moderate phenotypes, and are effective in species such as trees and polyploid grasses that cannot be readily bred to produce homozygous TILLING-based alleles. One approach is to modulate gene expression level, and pattern of expression, using carefully chosen or engineered promoters to target gene expression to defined developmental stages and tissue types. The success of this approach was demonstrated in rice, where tissue-specific expression of the *GA2ox* gene was able to impart a desired dwarf phenotype without affecting grain yields. The *OsGA2ox1* gene was placed under the promoter of an *OsGA3ox2* biosynthetic gene, preventing the undesired pleiotropic effects that had been observed with a promoter that imparted constitutive expression (Sakamoto *et al.*, 2003). The main limitations to this approach are three. (1) For most species, a set of 5′ regulatory regions that reliably impart diverse patterns of highly tissue-specific and/or cell-specific expression to transgenes have not been characterized. (2) The rules for promoter engineering are very poorly known; for many promoters it is even difficult to detect sequence homologies between promoters of genes that have orthologous sequences and gene expression patterns. (3) Because of the lack of an efficient system for gene targeting/homologous recombination in plants, even well-characterized promoters often give rise to widely varying patterns of expression among transformation events, requiring that large numbers of transgenic events are screened to find a usable, if not a perfectly genuine, pattern of expression. However, this inefficiency also provides an advantage in that it allows breeders to take advantage of this transformation-imparted variation in selecting for variable numbers of copies of gene insertions, or for variable levels and patterns of expression of single insertions. The former method was demonstrated in controlling tomato fruit size; different inserted numbers of copies of the *fw2.2* gene produced a continuum of correlated expression levels and fruit sizes (Liu *et al.*, 2003).

It was just two alleles in two agricultural species that played a major role in bringing about the Green Revolution (Silverstone & Sun, 2000). Armed with knowledge of the identities of these and many other genes with similar effects, and with rich and growing genomic databases in many plant species, the possibilities for new kinds of stature and form modification, and for fresh insights into the evolution of plant form, seem limitless.

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