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Genetic control of floral size and proportions

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ABSTRACT Floral size is an ecologically important trait related to pollination success and genetic fitness. Independently of the sexual reproduction strategy, in many plants, floral size seems to be controlled by several genetic programs that are to some extent independent of vegetative growth. Flower size seems to be governed by at least two independent mechanisms, one controlling floral architecture that affects organ number and a second one controlling floral organ size. Different organ-dependent growth control may account for the final proportions of a flower as a whole. Genes controlling floral organ identity, floral symmetry and organ polarity as well as auxin and gibberellin response, also play a role in establishing the final size and architecture of the flower. The final size of an organ seems to be controlled by a systemic signal that might in some cases overcome transgenic modifications of cell division and expansion. Nevertheless, modification of basic processes like cell wall deposition might produce important changes in the floral organs. The coordination of the direction of cell division and expansion by unknown mechanisms poses a challenge for future research.

KEY WORDS: *floral meristem, cell cycle, systemic signal, floral patterning, floral architecture*

The final shape and body size of multicellular organisms is the result of a genetic program and the influence of environmental conditions. In animals and plants the intrinsic growth rate is modulated by nutrient availability that determines the final size of the organism. In animals, both body size and longevity are to some extent controlled by the insulin pathway that is in itself dependent on nutrient conditions (Nijhout, 2003). But one important difference between plants and animals is that in plants, the formation of the different organs happens after embryonic development, thus not only organ or body size is influenced by environmental clues but also the types of organs produced. Our understanding of the way final plant size is achieved has been obtained using two different approaches: physiologists have tried to understand the roles of the so called plant growth regulators and environmental signals on plant development whereas geneticists have concentrated their efforts in finding mutants, genes or natural variation affecting growth in any of its forms. Although these two research lines appear separate, the reality is that they have been linked by an enormous amount of work done by plant breeders studying gene and environment interactions on agricultural traits that are related to growth, like yield, fruit size, biomass production etc. The efforts done in the model system *Arabidopsis* have helped to bring together the more basic approaches since mutations affected in plant growth regulator synthesis, degradation and the transduction of the signal have been isolated and characterized.

Which are the mechanisms that control the final size of an organism is a question without a clear answer yet. There are two basic processes that could contribute to its control: cell division and expansion but the integration of these two cellular programs into the context of organ growth and development is poorly understood (Torii, *et al.,* 1996). Plant growth and determination of the final size of plant organs is a modular process, happening throughout the entire lifespan in response to intrinsic developmental patterns and external conditions (Doonan, 2000). Both cell division and cell expansion contribute to organ growth and factors determining the integration of these two cellular processes into the context of organ growth and development are the topic of many investigations both in animals and plants (Day and Lawrence, 2000, Potter and Xu, 2001).

Despite the enormous amount of knowledge accumulated in *Arabidopsis* by cloning and genetic analysis of developmental mutants in most cases phenotypic descriptions of flowers tend to be scarce, suggesting that either some of the mutants have subtle floral phenotypes, or that the effect is too complex to be described in simple terms. In this review we discuss the recent advances in the understanding of the control of organ size and proportions with a special emphasis on floral size in different model systems.

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Ecological and evolutionary significance of floral size and proportions

Most plants can be roughly classified into two different reproductive strategies, those that largely rely on outcrossing with other members of the species (allogamy) and those that tend to fertilize themselves (autogamy). In allogamous species, the dependence in some cases on pollinators for reproduction has led to the development of ecological traits that have a function in attraction of birds, insects, bats etc. A detailed review about the evolution of color and related traits has been published recently by Clegg and Durbin (Clegg and Durbin, 2003). One of the traits that play a role in pollination of allogamous species is floral size. Floral size is studied under the so called mating system, since in many cases there is some kind of coevolution between floral size traits and pollinators. For instance, larger corollas seem to attract more pollinators and in this respect there might be an advantage for larger petals, but studies comparing hermaphroditic plants with gynodioecious plants (having either female or hermaphroditic plants) show that hermaphroditic plants tend to have smaller flowers probably due to preferential allocation of energy into fruit and seed formation (see Miller and Venables and references therein) (Miller and Venable, 2003). The general picture suggests that there is a strong genetic control over floral size and the relative growth of the organs, timing to anthesis and coincidence in time and space of fertile pollen and receptive stigma.

Most of the advances in our understanding of floral size and its control at the molecular level have come from *Arabidopsis* and *Antirrhinum*, two species with quite opposite reproduction strategies. *Arabidopsis* is a selfing annual (Somerville and Koornneef, 2002) that is partially cleistogamous whereas *Antirrhinum* in nature is a perennial and strictly allogamous (Lai *et al.,* 2002, Qiao *et al.,* 2004). The *Antirrhinum* research lines however are self pollinators (Xue *et al.,* 1996) due to mutation of the S-locus. This difference might be apparent in the future since it is currently difficult to predict the degree of conservation between genes controlling floral organ size in a plant like *Antirrhinum*, where floral size traits might be under strong evolutionary pressure and *Arabidopsis* where floral size might not play a major role in fertilization.

Fig. 1. Separation of vegetative and floral growth traits. *Pictures corresponding to F1 plants of a cross between* A.majus *and* A. linkianum *showing the third leaf and first flower of three different plants segregating floral size, but showing similar leaf size.*

Coupling between vegetative and reproductive organ size

Different degrees of coupling between floral and vegetative traits and among floral characters was already observed by Berg (Berg, 1959). In plants with specialised pollination systems, flower parameters were observed to vary less than vegetative traits with a tighter phenotypic integration of floral characters than in plants with unspecific mating systems. Later investigations found that a de-coupling of vegetative from floral traits is species specific and can also be found in plants with unspecialised pollination systems (Armbruster *et al.,* 1999). A tight coupling between floral and vegetative traits could be explained either by an environmental correlation, that means a common response to an environmental cue, or by genetic correlation, that means a common inheritance due to a pleiotropic effect of a single gene on a set of developmentally related traits (see below) or a linkage desequilibrium between separate genes with effect on different characters (Juenger *et al.,* 2000). An additional way to separate vegetative and floral gene functions is by allele specific interactions, recently shown in a *cincinnata* allele in *Antirrhinum* (Crawford *et al.,* 2004) (see below).

In *Antirrhinum*, we have been studying different mutations that affect foral size and proportions and we have found that the classic mutations *compacta, muscoides* (Stubbe, 1966) and *nanalargiflora* (Stubbe, 1974) and the newly identified *ktana* (Weiss and Egea-Cortines, unpublished), affect both vegetative and floral development whereas the classic mutants *compacta ähnlich, formosa, Grandiflora* and *Nitida* seem to affect only the flower under normal growth conditions. This suggests that at least two sets of genes that control floral size and proportions, one that has functions both in vegetative and reproductive growth and a second one that is probably flower specific. Natural variation is a great resource in *Antirrhinum* since there are more than 16 wild species that can be crossed with each other. In an F2 of *A. majus* 165E line (Sommer *et al.,* 1985) with the wild *A. majus.ssp linkianum* we have found segregation of floral size in plants that show nearly identical leaf size suggesting that there is a degree of separation between genes controlling leaf expansion and floral size (Fig. 1). The analysis of natural variation in a recombinant inbred line built from *A. majus* and the wild species *A. charidemi* shows that several QTL controlling floral size are specific for the flower, confirming a partial separation of vegetative and reproductive control (A. Hudson personal communication). A recent survey of QTL controlling leaf, sepal and petal size in tomato has shown that there is no overlap between QTL controlling the same trait in different organs (Frary *et al.,* 2004). These results seem to be also true in *Arabidopsis* where a comparison of QTL affecting leaf and floral development have found eleven QTL that affect only one of the organs and two that have pleiotropic effects (Juenger *et al.,* 2005). The partial isolation of the flower from the rest of the plant in terms of regulatory genes might be true even for some basic processes like sugar sensing, that is central to plant development (Leon and Sheen, 2003). For instance the loss of function of the glucose sensor hexokinase *glucose insensitive 2* (*gin2*) in *Arabidopsis* causes extreme dwarfism but floral size is apparently normal (Moore *et al.,* 2003).

From a developmental perspective, it is generally agreed that the activation of the major switch that causes the shoot apical

meristem (SAM) to produce floral primordia instead of leaves or branches is the *FLORICAULA* (*FLO*) gene in *Antirrhinum* (Coen *et al.,* 1990), *LEAFY* (*LFY*) in *Arabidopsis* (Weigel *et al.,* 1992), *FALSIFLORA* in tomato (Molinero-Rosales *et al.,* 1999) or *ABERRANT LEAF NON FLOWER* in *Petunia* (Souer *et al.,* 1998). Ectopic expression of *LFY* or its orthologs in the shoot apical meristem causes increased flowering speed in many systems like *Arabidopsis*, poplar (Weigel and Nilsson, 1995) or orange trees (Pena *et al.,* 2001), but does not cause ectopic floral tissues to develop outside floral primordia. This suggests that the floral context, understood as the specific transcriptome and proteome that leads to the formation of inflorescence primordia, is largely, but not completely, controlled by the *FLO/LFY* switch. Indeed in some plants like tobacco or vine, the *FLO/LFY* ortholog is expressed in vegetative meristems suggesting an evolutionary divergence (Carmona *et al.,* 2002, Kelly *et al.,* 1995). Thus it is formally possible that the observed genetic separation between floral and vegetative traits might be a genetic program activated by the floral context, or is an intrinsic part of it in those species that have this characteristic.

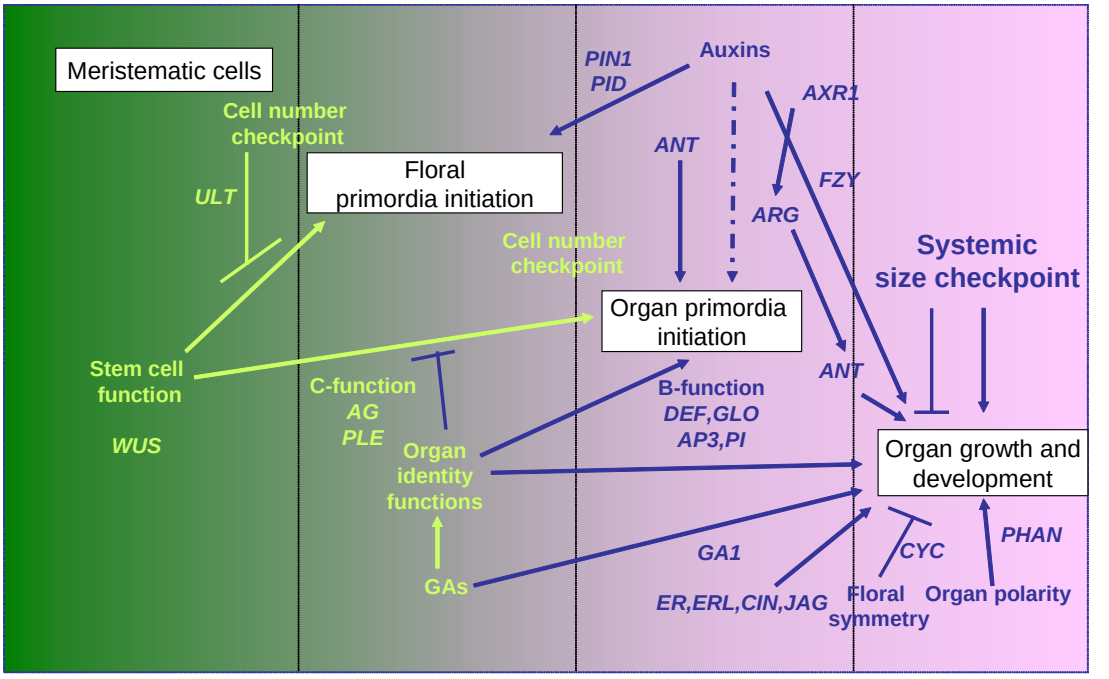

Fig. 2. A model describing the events and some of the genes known to affect floral size and proportions.

Genes controlling floral size and architecture

The control of floral size can be separated into two different aspects, one is the control of the number of organs in a whorl, thus affecting floral size in terms of sheer number of organs and a different mechanism is that controlling the size of each of the organs formed within a flower. The flower, like the rest of the organs of higher organisms, has a certain “normal size” in a species. During plant development, after transition from vegetative to reproductive growth, the SAM produces flowers instead of leaves and there are different sets of genes that regulate cellular mechanisms during each developmental step. As floral development proceeds, local regions of cell division establish individual floral organ primordia at specific distances and angles from each other. This process is controlled by three classes of genes, those that affect development of floral primordia, those that alter floral symmetry and those that specify organ identity (Schwarz-Sommer *et al.,* 1990). Cell number in the centre of the meristem of *Arabidopsis* is, among others, regulated by the genes *CLV* and *WUS* (Clark *et al.,* 1997, Haecker *et al.,* 2001, Irish and Jenik, 2001, Kayes and Clark, 1998, Laufs *et al.,* 1998, Mayer *et al.,* 1998). The cell number in floral primordia, the spacing of organ inception, the determination of organ shape and the specification of organ size are all controlled separately (Meyerowitz, 1997). A proposed model of determination of floral size based on the data described below and our own observations is shown in Fig. 2. A list with some of the genes controlling floral size and proportions can be seen in Table 1.

Mutants that affect the number of floral organs include *PERIANTHIA (PAN)* (Chuang *et al.,* 1999, Running and Meyerowitz, 1996), *ETTIN (ETT)* (Sessions *et al.,* 1997), *WIGGUM* (*WIG*) (Running *et al.,* 1998) or the *SUPERMAN* gene of *Arabidopsis* (Huang and Ma, 1997, Jacobsen and Meyerowitz, 1997). The mutants *pan* and *ett* have in common an increase in sepals and petals and decrease in stamen numbers whereas *sup* produces more stamens at the expense of carpels. In contrast *wig* has more organs in all the whorls and shows synergistic interactions with other genes controlling cell division in primordia like *CLV*.

Gene mutations which control the overproliferation of cells in the SAM like the three *CLAVATA* genes show similar phenotypes with increased organ number in the flower and modified floral architecture (Brand *et al.,* 2000, Clark *et al.,* 1993, Clark *et al.,* 1997, Fletcher *et al.,* 1999). The size of the floral anlage seems to play a role in the number of organs that are formed since several genes are involved in restricting the accumulation of cells in floral primordia. For instance the *ULTRAPETALA* (*ULT*) mutation has more organs than wild type (Fletcher, 2001) and it seems to control the size of the floral meristem through control of *CLV* expression and repression of *WUS* activity (Carles *et al.,* 2004).

A large number of mutants affect floral architecture via effects on general cell division, meristem size, or cell allocation to primordia. Plants affected in the *STRUWELPETER* gene (*SWP*) show smaller organs in the aerial part of the plant and reduced organ number in the flower, leading to abnormal architecture (Autran *et al.,* 2002). Genes involved in stem cell formation like

wuschel (Laux *et al.,* 1996; Mayer *et al.,* 1998) also show floral phenotypes that include a decrease in the number of organs in the flower that seems to be random. Organ number phenotypes tend to be stronger in stronger alleles. All these mutants have in common a decrease in the number of cells that form floral primordia suggesting that there is a minimal threshold of cells required to form an organ and below this critical number, floral architecture is compromised.

Floral homeotic genes

The observation of many of the classical floral homeotic mutants in *Antirrhinum* and *Arabidopsis* has led to the conclusion that all the major genes involved in the establishment of floral organ identity exert some control over growth in the flower in one way or another. Thus the floral organ identity genes *DEFICIENS-APETALA3* (Jack *et al.,* 1992; Sommer *et al.,* 1990), *GLOBOSA-PISTILLATA* (Goto and Meyerowitz, 1994; Trobner *et al.,* 1992) and *PLENA-AGAMOUS* (Bradley *et al.,* 1993; Yanofsky *et al.,* 1990) from *Antirrhinum* and *Arabidopsis* show effects on floral organ size.

Weak alleles of the B function gene *deficiens* like *defnicotianoides* or *defchlorantha* show decreased petal development and *defnic* stamens are shorter than in wild type (Sommer *et al.,* 1990) (see Fig. 3A). The temperature sensitive allele *def101* shows petals close to wild type organ size at 15ºC as compared to 25ºC when petal organs are absent and transformed into sepals (Zachgo *et al.,* 1995). Thus, although the primary function of B

TABLE 1

GENES AFFECTING FLORAL SIZE AND ARCHITECTURE IN DICOTYLEDONEOUS

Effect	Gene	Species	Reference
Floral Architecture			
Increased organ number			
	CLAVATA 1, 2 and *3*	*Arabidopsis*	(Clark *et al.,* 1993; Fletcher *et al.,* 1999; Kayes and Clark, 1998)
	ULTRAPETALA	*Arabidopsis*	(Fletcher, 2001)
	WIGGUM	*Arabidopsis*	(Running *et al.,* 1998)
	CYCLOIDEA	*Antirrhinum*	(Luo *et al.,* 1996)
	JAGGED	*Arabidopsis*	(Dinneny *et al.,* 2004; Ohno *et al.,* 2004)
Increased organ number- control of C function			
	AGAMOUS,PLENA	*Arabidopsis*, *Antirrhinum*	(Bowman *et al.,* 1989; Bradley *et al.,* 1993)
	POLYPETALA	*Antirrhinum*	(McSteen *et al.,* 1998)
Change in relative organ numbers			
	PLURIPETALA	*Arabidopsis*	(Running *et al.,* 2004)
	SUPERMAN	*Arabidopsis*	(Huang and Ma, 1997; Jacobsen and Meyerowitz, 1997)
	PERIANTHIA	*Arabidopsis*	(Chuang *et al.,* 1999; Running and Meyerowitz, 1996)
	ETTIN	*Arabidopsis*	(Sessions *et al.,* 1997)
Decrease in floral organ number			
	WUSCHEL	*Arabidopsis*	(Laux, *et al.,* 1996; Mayer *et al.,* 1998)
	STRUWELPETER	*Arabidopsis*	(Autran *et al.,* 2002)
	DEFICIENS	*Antirrhinum*	(Sommer *et al.,* 1990)
	LEUNIG	*Arabidopsis*	(Liu and Meyerowitz, 1995)
	STERILE APETALA	*Arabidopsis*	(Byzova *et al.,* 1999)
	APETALA2	*Arabidopsis*	(Crone and Lord, 1994; Kunst *et al.,* 1989; Maes *et al.,* 1999)
	PIN FORMED-1	*Arabidopsis*	(Bennett *et al.,* 1995, Okada *et al.,* 1991)
	PINOID	*Arabidopsis*	(Bennett *et al.,* 1995)
	FLOOZY	Petunia	(Tobeña-Santamaria *et al.,* 2002)
	AINTEGUMENTA	*Arabidopsis*	(Elliott *et al.,* 1996; Klucher *et al.,* 1996)
	FILAMENTOUS FLOWER	*Arabidopsis*	(Chen *et al.,* 1999; Sawa *et al.,* 1999)
	SKP	*Arabidopsis*	(Ni *et al.,* 2004)
	UNUSUAL FLORAL ORGANS	*Arabidopsis*	(Durfee *et al.,* 2003)
Increased floral organ size			
	FORMOSA	*Antirrhinum*	(Benarroch *et al.,* unpublished)
	GRANDIFLORA	*Antirrhinum*	(Benarroch *et al.,* unpublished)
	SPLENDIDA	*Antirrhinum*	(Benarroch *et al.,* unpublished)
	35S::*ARGOS*	*Arabidopsis*	(Hu*et al.,* 2003)
	35S::*AINTEGUMENTA*	*Arabidopsis*	(Krizek, 1999; Mizukami and Fischer, 2000)
	35S::UFO	*Arabidopsis*	(Lee *et al.,* 1997)
Decreased floral organ size			
	LIPLESS	*Antirrhinum*	(Keck *et al.,* 2003)
	CINCINNATA	*Antirrhinum*	(Crawford *et al.,* 2004)
	ERECTA and ERECTA-LIKE	*Arabidopsis*	(Shpak *et al.,* 2004)
	AtBRAHMA	*Arabidopsis*	(Farrona *et al.,* 2004)
	ECTOPIC LIGNIFICATION 1	*Arabidopsis*	(Caño-Delgado *et al.,* 2000)
	FRAGILE FIBERS 2	*Arabidopsis*	(Burk and Ye, 2002)
	EXPANSIN	Petunia	(Zenoni *et al.,* 2004)
	GIBERELLIC ACID 1	*Arabidopsis*	(Olszewski *et al.,* 2002)
	AUXIN RESISTANT1	*Arabidopsis*	(Leyser *et al.,* 1993)
	KTANA	*Antirrhinum*	(Benarroch *et al.,* unpublished)
	NITIDA	*Antirrhinum*	(Benarroch *et al.,* unpublished)
Floral organ proportions			
	COMPACTA	*Antirrhinum*	(Benarroch *et al.,* unpublished)
	COMPACTA ÁHNLICH	*Antirrhinum*	(Benarroch *et al.,* unpublished)
	UNILABIATA	*Antirrhinum*	(Benarroch *et al.,* unpublished)
	OVATE	Tomato	(Liu *et al.,* 2002)

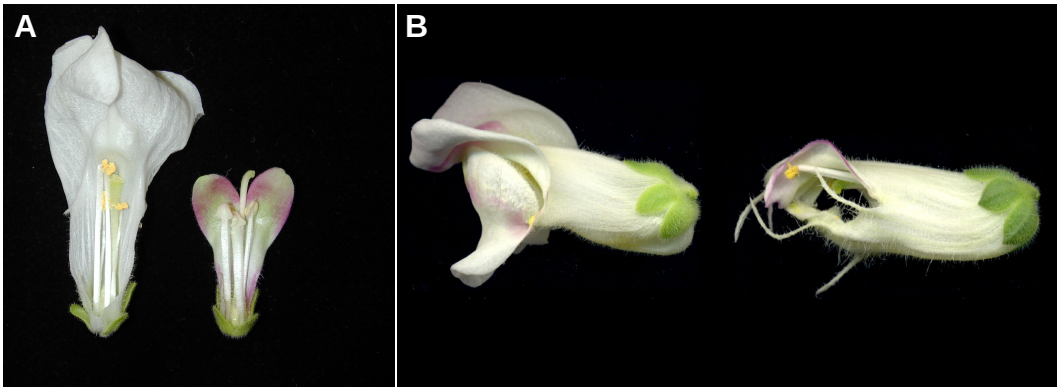

Fig. 3. Effect of homeotic and adaxial/abaxial patterning genes on floral size. (A) *Comparison between wild type (left) and* deficiensnicotianoides *flowers (right).* **(B)** *Wild type (left) and* phantastica *(right) (courtesy of R. Waites).*

function genes is the control of organ identity, they also play a role in activating petal and stamen growth. Loss of B function genes in *Arabidopsis* causes loss of organs in the third whorl for instance in *pi-1* (Bowman *et al.,* 1991) and the ectopic expression of *AP3·* and *PI* leads to the rescue or organ number in class A mutants and an increased number of stamens due to increased whorl number (Krizek and Meyerowitz, 1996) suggesting that B function genes play a dual role in controlling cell proliferation both in the formation of primordia and in organ growth in *Arabidopsis*. The activation of both B and C function genes is controlled by the F-box protein *FIMBRIATA* (*Fim*) (Ingram *et al.,* 1997; Simon *et al.,* 1994) and its *Arabidopsis* ortholog *UNUSUAL FLORAL ORGANS* (*Ufo*) (Ingram *et al.,* 1995; Levin and Meyerowitz, 1995; Wilkinson and Haughn, 1995). Ectopic expression of UFO in *Arabidopsis* causes increased floral organ size (Lee *et al.,* 1997) due to increased cell division (Mizukami, 2001). The fact that ectopic expression of B function genes does not cause an increase in floral organ size suggests that the increase resulting from UFO misexpression is not due to the higher B function activity observed, but to other unknown factors. This would be in agreement with the results of Ingram *et al.,* that suggest that *FIM* acts by selective degradation of regulatory proteins involved in controlling floral homeotic gene functions and cell division (Ingram *et al.,* 1997). Proteins of the F-box family can bind proteins of the SKP family and form complexes with target proteins that are degraded by the ubiquitin pathway (Ciechanover, A. 1998). Recent experiments show that loss of function of the *UFO* partners also cause major disruption of floral development including loss of organs and arrested petal development (Ni *et al.,* 2004).

The C function genes are involved in controlling organ identity and meristem determinacy in *Antirrhinum* (Bradley *et al.,* 1993), *Arabidopsis*,(Yanofsky *et al.,* 1990) tomato, (Pnueli *et al.,* 1994), petunia and cucumber (Kater *et al.,* 1998). Loss of C function activity results in formation of additional whorls in the inner part of the flower, substituting the carpels with a reiteration of sepal, petal, petal whorls in *Antirrhinum* and *Arabidopsis* (Davies *et al.,* 1999). Reduced activity of genes that are required to activate or maintain C function like *POLYPETALA,* also show increased organ number due to additional whorls of petals (McSteen *et al.,* 1998). This increase in organ number is caused by the maintainance of *WUS* in the floral meristems that in normal conditions would be repressed by the C function (Lenhard *et al.,* 2001; Lohmann *et al.,* 2001). The genetic analysis of *CLV, ULT, WUS* and *AG* show that *WUS* plays an indirect but important role in establishing the number of cells available to form floral primordia at early stages, before its inactivation by C function to terminate the flower.

The classical *Arabidopsis* A function genes controlling floral organ identity like *AP2* (Crone and Lord, 1994; Kunst *et al.,* 1989; Maes *et al.,* 1999) or other repressors of C function expression in the perianth like *LEUNIG* (Liu and Meyerowitz, 1995) or *STERILE APETALA* (Byzova *et al.,* 1999) have strong effects in the architecture of the flower. Ectopic expression of the *Arabidopsis AP2* in *Petunia* causes flowers with increased organ number (Maes *et al.,* 1999). Since the A function is a negative regulator of the expression of *AGAMOUS* and *AG* is known to inhibit *WUS* (Lohmann *et al.,* 2001), it is formally possible that the observed loss of organs in A function mutants is caused by the premature repression of *WUS* by ectopic *AG* expression. Similarly the increase in organ number by ectopic *AP2* expression could result from inhibition of *AG* and therefore increased *WUS* activity.

In spite of considerable knowledge of the function of these genes in *Arabidopsis*, the situation seems to be different in *Antirrhinum* and Petunia. *Petunia* has three AP2-like genes, one of them can complement the *Arabidopsis* mutation and the existence of three paralogs has limited tests of functional conservation (Maes *et al.,* 2001). Furthermore the two *Antirrhinum AP2* orthologs, known as *LIPLESS* do not show loss of organ number in double mutant combinations (Keck *et al.,* 2003) (see below).

Auxins and floral development

Auxins play an important role in lateral organ formation and early floral development (Bennett *et al.,* 1995; Okada *et al.,* 1991). Detailed analysis of mutations that resemble *Arabidopsis* plants grown in the presence of auxin transport inhibitors suggest that the so-called *pin* group of mutations, generally affected in lateral organ primordia initiation and floral patterning, are in fact mutants affected in auxin transport and signalling. Mutations in *PIN1* or *PID* develop flowers that display a decrease in the number of sepals and stamens and an increase in petals suggesting that *PID* plays a role in the formation of organ primordia (Bennett *et al.,* 1995). The overexpression of PID causes a strong defect in patterning including lack of lateral organs that is thought to be the result of a shift of apical-basal targeting of the PIN1 protein (Friml *et al.,* 2004). Recent work has identified auxin synthesis genes like the *Yucca* gene from *Arabidopsis* that encodes a flavin monooxygenase (Zhao *et al.,* 2001). Its Petunia ortholog, *FLOOZY* (*FZY*), plays a major role in floral architecture since *fzy* mutants lack in most cases the organs of the outer three whorls (Tobeña-

Santamaria *et al.,* 2002). However it is interesting that in *fzy* mutants SEM analysis of the floral meristems show that organ patterning is correct and the lack of organs is due to a failure of the different primordia to grow. Although the current data support the hypothesis that auxins play a major role in primordia initiation (Reinhardt *et al.,* 2003, Reinhardt *et al.,* 2000), the results suggest that in flower development auxins might be dispensable for the initiation of floral primordia, or in *fzy* mutants there is a mechanism that allows enough auxin to accumulate to produce primordial but not to maintain organ growth.

The data from the above genes show that the total number of cells allocated to form floral primordia is important to maintain floral architecture. Since each organ seems to achieve more or less wild type size in many mutants affected in floral architecture, the number of cells allocated to form floral organ primordia might be maintained in the mutants and a deficit or excess of cells translated into a modified number of organs. All together, these data suggest that cell number is a factor controlling organ size since an unknown mechanism seems to allocate a minimum amount of cells to each primordium that ensures proper organ formation. In those cases where cell division after primordia initiation is also compromised, organ size might also be affected (see below).

Genes that affect floral symmetry

One of the main differences in floral architecture in angiosperms is the existence of two main kinds of flowers, those with a zygomorphic symmetry like *Antirrhinum* and those with radial symmetry like *Arabidopsis*, Petunia or tomato (Coen and Nugent, 1994). Pioneering work in *Antirrhinum* has shown that the genes involved in establishment of the zygomorphic symmetry like *CYCLOIDEA* (*CYC*) (Luo *et al.,* 1996) or *DICHOTOMA* (Luo *et al.,* 1999) belong to the TCP family of transcription factors (Cubas *et al.,* 1999). Mutants in the *CYC* locus have five stamens since the adaxial stamen that in wild type aborts grows to normal size. *In situ* hybridization experiments show that *CYC* inhibits the cell cycle genes *CYCLIN D3b* and *HISTONE H4* in the position of the staminoids whereas in *cyc* mutant plants, expression of the cell cycle genes predicts formation of a functional organ (Gaudin *et al.,* 2000). Altogether these data confirm the general idea that organs cannot form without cell division.

Genes that affect floral organ size

A way to analyse the regulation of organ size is by studying the pattern of cell division and expansion during development and how they are affected in mutants that show modifications in leaves and/or flowers (Tsuge *et al.,* 1996). Studies of mutations with pleiotropic effects frequently describe morphological or cellular changes in the leaves, whereas the effect on other shoot organs, especially the different floral organs, is treated less profoundly (Hu *et al.,* 2003; Smith *et al.,* 1996; Tsuge *et al.,* 1996; Tsukaya, 2003; Wyrzykowska *et al.,* 2002; Wyrzykowska and Fleming, 2003). We review below some of the genes for which detailed descriptions of floral phenotypes are available.

The *AINTEGUMENTA* mutation was isolated independently by two groups as a female sterile mutation in *Arabidopsis* (Elliott *et al.,* 1996; Klucher *et al.,* 1996). The *ANT* locus encodes a transcription factor of the AP2 family and the loss of function of the gene causes severe disruption of ovule development. The defects include lack of integument, a block of embryo sac formation and arrest of megagametogenesis. The development of the floral organs is also strongly affected, showing random loss of organs in the outer three whorls and a strong effect on organ growth. Amongst the phenotypes described, the *ant-9* allele shows serrated sepals, narrow petals, thin anthers, sepalloid and petalloid stamens and unfused gynoecium (Elliott *et al.,* 1996), whereas *ant-1* shows similar phenotypes but petals width is more often affected than length (Klucher *et al.,* 1996). *AINTEGUMENTA* is also expressed in vegetative primordia (Elliott *et al.,* 1996) and roots (Klucher *et al.,* 1996) and plants homozygote for *ant-1* have smaller leaves than wild-type. The random losses of organs in *ant* mutants suggest that the general cell division process is severely disrupted and, as we proposed above, both floral anlagen and further organ development seem to be affected. The *ant* gene has an additional function promoting petal identity that might also explain the decrease in petal size in *ant* mutants (Krizek *et al.,* 2000).

The overexpression of *ANT* in *Arabidopsis* causes increase in organ size in the flower. The increase in organ size is due either to increased cell division in sepals and cell expansion in the inner three whorls (Krizek, 1999), or to increased cell division in petals too (Mizukami and Fischer, 2000). Mizukami and Fischer also found increased vegetative growth. Plants overexpressing *ANT* produce larger leaves as the result of an extended period of leaf growth caused by a longer period of cell division. Consistent with this observation, cyclin D3 and histone H4 were found to be expressed ectopically in sepals of 35S::ANT flowers. *AINTEGUMENTA* is thought to promote growth at different levels, both in the shoot apical meristem as well as within floral primordial and in floral organs. The results discussed above suggest that both rate and duration of cell division are important to determine the final size of both lateral and floral organs.

Auxins play a role in establishment of primordia and expansion of lateral organs. The gene *ARGOS* was identified in a microarray experiment studying the effect of the auxin naphthylacetic acid (NAA) on roots of 7-day old *Arabidopsis* plants (Hu *et al.,* 2003). Its expression is not confined to roots and it can be detected in aerial parts of the plant, stems, rossete and flowers. The cellular localization of the GFP fusion shows a general distribution of the protein throughout the cell, but the molecular function of the protein remains to be established. Manipulation of *ARGOS* gene expression by antisense and overexpression shows that it plays a role in general organ growth since antisense plants have smaller organs in the aerial parts, including leaves, flowers and fruits whereas plants with increased *ARGOS* mRNA have larger leaves, flowers and siliques. Moreover, the overall plant size corresponds to the single organ phenotypes suggesting that *ARGOS* may act in the general signal transduction pathway of auxins involved in growth control. A detailed analysis of 35S::*ARGOS* plants shows that the increased size of the organs is due to an extended period of cell division and not an increase in growth rate. One possible explanation for this phenomenon is the observed overexpression of *ANT* in 35::*ARGOS*, that suggests that *ARGOS* acts via activation of the *ANT* gene. This hypothesis seems to be correct since the increased growth observed in 35S::*ARGOS* is abolished in an *ant-1* genetic

background. *ARGOS* acts downstream of some auxin genes like *AUXIN RESISTANT AXR1* since 35S::ARGOS can partially restore organ development in *axr1* mutants, suggesting that auxins may act in promoting general growth via *ARGOS* and *ANT* defining the timing of cell division in all aerial primordia.

The classical mutation *erecta* has a compact inflorescence, round leaves with short petioles and short and round siliques (Torii *et al.,* 1996). The *erecta* mutation has no effect on floral organs, but this is due to genetic redundancy with *ERECTA* -like genes. The *ERECTA* gene codes for a receptor-like kinase and there are at least seven genes similar to *ERECTA* and over 600 receptor kinases in the *Arabidopsis* genome. Loss of function alleles in the two *ERECTA-LIKE* genes (*ERL1* and *ERL2*) do not show an apparent phenotype, but double mutants with *er* enhance some of the aspects of the mutation like shorter siliques and pedicels for the *erl1-2, er105* double mutant and shorter internodal elongation in *erl2-1 er105* double mutants (Shpak *et al.,* 2004). Interestingly the triple mutant *er105, erl1-2, erl2-1* has strong phenotypes over all the aerial organs. Flowers of the triple mutant lack pedicel and floral organs and those that have inner organs, show small needle petals, small anthers and very short gynoecium. The *erecta* mutation has been found to cosegregate with QTL affecting floral size suggesting its importance in the overall control of floral size in *Arabidopsis* (Juenger *et al.,* 2000). All together these data suggest that *ER* like *ANT* or *ARGOS* affect general growth by transducing signals from plant growth regulators, or from other cells that are integrated into organ growth and development.

The *CINCINNATA* mutant of *Antirrhinum* was identified by Stubbe in 1932 (Stubbe, 1932) and described as pleiotropic (Stubbe, 1966). Identification of the *CIN* sequence resulted from inactivation of a gene belonging to the TCP family of transcription factors (Cubas *et al.,* 1999; Nath *et al.,* 2003), whose loss of function phenotype is similar to the mutants *CIN* and *SUBCRISPA*. These mutants are allelic and have strong phenotypes both in leaves and flowers, but in contrast to *ANT* or *ARGOS*, *CIN* is not controlling general cell proliferation in leaves or flowers, since the proposed mode of action of *CIN* is to allow cell division to arrest in response to extracellular signals in the leaves and promote cell division in petal lobes in the flower (Crawford *et al.,* 2004). *CINCINNATA* also affects the development of conical cells both in leaves and petals, a process that is controlled by the *MIXTA* gene in petals (Noda *et al.,* 1994), suggesting that *CIN* has several biological functions. One interesting aspect of the work of Crawford *et al.,* is that the weak allele *cin-628* has an effect on conical cells but has no effect on leaf development suggesting that floral and leaf development could be genetically separated in the case of genes that have strong effects on leaf morphology. Recent work shows that the *JAW* locus of *Arabidopsis* that display phenotypes similar to those described for leaves mutated in *CIN* encodes a microRNA that controls TCP4, a TCP gene closely related to *CIN*, suggesting that the overall control over *CIN* and orthologs is important in plant morphogenesis (Palatnik *et al.,* 2003).

The *JAGGED* gene in *Arabidopsis* has been isolated independently by two groups (Dinneny *et al.,* 2004; Ohno *et al.,* 2004). Loss of function of *JAG*, causes a slight modification of floral architecture with increase in floral organ numbers in the perianth but stamen and carpel numbers are similar to wild type (Ohno *et al.,* 2004). Petals in plants homozygous for *jag* are shorter than the wild type and are serrated in the distal part (thus the name of the gene) and a weak serration can be seen in leaves. Studies of cell size (Ohno *et al.,* 2004) and distribution of dividing cells using *in situ* hybridization (Dinneny *et al.,* 2004a), show that both cell number and shape are affected in the mutant. The *JAG* gene seems to be required to promote growth of lateral organs and the coincidental expression of *JAG* with *GUS* driven by the *Cyclin1At* promoter suggests that *Jag* might be necessary to maintain cell division in the margins of organs. The *ANT* and *ARG* genes are also involved in maintenance but in contrast to *ANT* or *ARG*, *JAG* overexpression does not cause increased organ size and the main phenotype seen is the development of leaf organs instead of flowers, suggesting that *JAG* and *ANT* do not share target genes.

An additional clue to the compartment hypothesis of floral organ development is given by the *LIPLESS* genes that were identified in *Antirrhinum* using a reverse genetics approach to study the orthologs of the *Arabidopsis* A function gene *APETALA2* (Coen and Meyerowitz, 1991) (Jofuku *et al.,* 1994) (Keck *et al.,* 2003). *APETALA2* is a member of a large family of transcription factor genes (Okamuro *et al.,* 1997). *Antirrhinum* has two genes with close homology to AP2 and only *lip1,lip2* double mutants show a novel phenotype in which the lips of the *Antirrhinum* flower fail to develop. The petal cells showed a morphology that is somewhat different from the regular flat cells present in the proximal part of the lips or the conical cells of the distal part (Glover *et al.,* 1998, Noda *et al.,* 1994). Petal growth is strongly reduced and reduction of the inner organs include shorter stamens and style, but the ovary is twice the length of the wild type (Keck *et al.,* 2003). In contrast to the defects in growth of petal, stamen and style, the sepals of *lip1,lip2* double mutants are larger than the wild type and have glands typical of bracts and leaves suggesting that *lip1* and *lip2* share a function in repressing some vegetative aspects that in a sepal context lead to increased organ size. Indeed the *lip, lip2, def* and the *lip, lip2, ple* triple mutants show that the organ effects are specific of each organ identity.

In terms of floral size and organ expansion, there is a large body of evidence supporting a key role of gibberellic acid (GAs). Many mutants affected in GA synthesis have underdeveloped floral organs (Olszewski *et al.,* 2002). The genes involved in repression of GA signalling belong to the DELLA family of proteins and a decrease in the function of the protein family progressively restores stamen development in the strong gibberellin synthesis mutant *GA1* (Cheng *et al.,* 2004). Recent work shows that the floral homeotic genes *AP3, PI* and *AG* are directly activated by GA during flower development and using inducible promoters Yu *et al.,* have shown that the homeotic genes are downstream of GA signalling (Yu *et al.,* 2004).

Genes controlling polar organ growth

Pioneering work in *Antirrhinum* led the foundation of our current understanding of leaf development with the identification of a genetic program controlling dorsal/ventral (adaxial/abaxial) symmetry in lateral plant organs (Waites *et al.,* 1998; Waites and Hudson, 1995). The *PHANTASTICA* gene product is a Myb transcription factor expressed throughout leaf primordia that establishes organ polarity. *PHANTASTICA* has functions in floral

development that include promotion of petal lobe development (Waites and Hudson, 2001) (see Fig. 3B) where adaxial/abaxial identity of the tissue is established. Phenotypic and genetic analysis of the *HANDELBARS* gene of *Antirrhinum* suggest that dorsal ventral asymmetry in floral organs might share components of the dorsal ventral pathway from leaves. Further genes involved in this process include the YABBY family of transcription factors (Bowman *et al.,* 2002) and HD-ZIPIII (Class III homeobox/leucine zipper) like *REVOLUTA*, *PHABULOSA* and *PHAVOLUTA* (see Hay *et al.,* for a recent review) (Hay *et al.,* 2004). Although most of the work on genes controlling establishment of organ polarity have been done in characterization of the leaf, some of the genes belonging to the YABBY family have floral phenotypes related to the subject of this review. The loss of function of the *FILAMENTOUS FLOWER* (*FIL*) gene causes loss of sepals and petals and substitution of stamens by filaments (Sawa *et al.,* 1999). The lack of organ number in *fil* has been interpreted to be caused by the ectopic expression of some of the floral organ identity genes (Chen *et al.,* 1999), suggesting that YABBY genes play a role in controlling patterning not only in the leaf but also in the flower. The ectopic expression of *FIL* does not cause increased floral organ number (Siegfried *et al.,* 1999), but YABBY genes may play a general role in organ size development since expression of YABBY3 from the *KANADI* promoter produces giant flowers (Yuval Eshed, personal communication). This result would be in agreement with the model proposed by Chen *et al.,* that maintains that the establishment of a proximo-distal axis controlled by YABBY genes might be important for organ expansion. This hypothesis is also favoured in maize where the mutant *rolled leaf* has been found to be important for lateral outgrowth of the leaf (Juarez *et al.,* 2004).

Fig. 4. Mutations affecting floral organ proportions. (A) *Comparison between wild type (left) and* compacta *(right).* **(B)** *Comparison between wild type (left) and* compacta ähnlich *(right).*

In *Antirrhinum* the *GRAMINIFOLIA* (*GRAM*) gene promotes lateral growth of the leaf and has a subtle floral phenotype (Golz *et al.,* 2004). Genetic and molecular interactions between *GRAM* and the gene controlling C function *STYLOSA* (*STY*) (Motte *et al.,* 1998; Navarro *et al.,* 2004), show that *GRAM* is involved in whorl positioning in the flower redundantly with *STY.*

STYLOSA encodes an ortholog of the Drosphila/yeast GRO/TUP1 corepressor and the *Arabidopsis* gene *LEUNIG* (Conner and Liu, 2000) and seems to be affected in hormone mediated processes that affect both leaf and floral organs (Cnops *et al.,* 2004; Navarro, 2004). *LEUNIG* and *AINTEGUMENTA* have been found to play a role in repression of C function and activation of cell division that supports marginal tissue development (Liu *et al.,* 2000). All together this complex networks of genetic interactions suggest that genes controlling proximo-distal or adaxial-abaxial development interact with other genes that are required to establish identity boundaries and cell proliferation in a genetic network that is not yet very well understood.

Organ proportions

The maintenance of floral proportions is of primary importance in plants since both autogamous and allogamous species require the right positioning of the sexual organs to ensure either self pollination or pollen dispersal and the perianth plays a vital role ensuring the microclimate of humidity and protection against environmental factors required for fertilization. The literature provides several examples showing that not all the organs of a plant respond equally to disruption or modification of the cell cycle with the cell cycle machinery or cell expansion being affected depending on the cellular context. The expression of a *CDC2* mutant in tobacco for example, which results in a cell cycle retardation, produces leaves of normal size with fewer, but bigger cells, whereas flowers and seeds contained fewer cells but of normal size. Whereas in the leaves the reduced proliferation was compensated by cell expansion, nuclear division cycle and cell expansion in flowers and seeds seemed to be more tightly coupled, (Hemerly *et al.,* 1995). Similarly, ectopic expression of *ANT*, which results in bigger organs, causes an increase in cell division in the sepals, whereas the increased sizes in petals, stamens and carpels are primarily attributable to an elevated cell expansion (Krizek, 1999). Another example is the *OVATE* gene that represses growth in tomato fruits (Liu *et al.,* 2002), since additional copies of the functional gene lead to a decrease in overall aerial growth including growth of leaves and floral organs. However sepals and stamens are more affected by the dosage effect of *OVATE* than petals and styles suggesting an interaction between the ovate gene product and the organ identity context.

We have been studying mutations specifically affected in floral proportions in *Antirrhinum* and the results show that at least perianth and sexual organ development, petal tube and petal lobes can be genetically separated in *COMPACTA* (Fig. 4A), *COMPACTA ÄHNLICH* (Fig. 4B) and *UNILABIATA* (Delgado-Benarroch, Weiss and Egea-Cortines unpublished results).

What limited data is available on this topic clearly suggests a differential growth response of each floral organ that is

intrinsic to its identity and that allows the proper floral proportions to be achieved, apparently by interaction of several developmental programs, some of them still to be identified.

Control of organ size by cell division and expansion

Mutant analysis and transformation experiments in plants show that changes in organ size can be traced back to an increase or decrease in either cell number or cell expansion or a combination of both, although changes in cell proliferation do not always correlate with changes in organ size because intrinsic mechanisms seem to coordinate cell proliferation and growth (Meyerowitz, 1997; Shpak *et al.,* 2003).

Cell division is regulated by the cell cycle machinery and a set of key transitions regulate the entry and progress through the cycle (G0-G1, G1-S and G2-mitosis). Transitions are controlled by the activity of cyclin-dependent protein kinases (CDKs) and these are typically activated by the synthesis of cyclins, other interacting proteins and the reversible phosphorylation of key amino-acid residues (Doonan, 2000). There are examples of a direct correlation between cell cycle and organ size variation (see examples above). On the other hand many works show that a variation in cell number does not cause variation in organ size. This is seen when *CDC2* is down regulated in transgenic tobacco plants, which produce normal sized leaves with less but larger cells (Hemerly *et al.,* 1995). Similarly, when cell division in young wheat leaves was blocked by gamma irradiation, leaf growth and morphogenesis continued and cell size increased compared to the non-irradiated control (Haber, 1962). Later studies showed that manipulation of cell division at the whole plant level might not modify organ size. However there are several reviews that explain these results (Meijer and Murray, 2001). Examples showing that cell division indeed plays a role in organ size include mutants with increased or decreased organ size attributable to changes in cell number. For instance in the mutant of *REVOLUTA/INTERFASCICULAR FIBERLESS1* of *Arabidopsis*., growth and cell proliferation is prolonged, resulting in larger leaves and flowers and a bigger stem (Talbert *et al.,* 1995). On the other hand the *auxin resistant 1* mutant (*axr1)* has smaller leaves, inflorescence stems and floral organs caused by a decrease in cell number (Lincoln *et al.,* 1990). As the number of characterized mutants increases a general conclusion is that increases or decreases in organ size tend to be linked to modifications of the cell cycle, in terms of duration of proliferation, or to modifications in cell division capacity and potential.

Even though changes in cell expansion can be compensated to keep normal shape and size of an organ, some mutations that directly affect cell expansion show clear organ size phenotypes. The *Arabidopsis ROTUNDIFOLIA* (*ROT*) gene encodes a cytochrome P450 which appears to function specifically in polar elongation of leaf cells in the leaf-length direction (Kim *et al.,* 1998) and the *ANGUSTIFOLIA* (*AN*) gene which regulates polar elongation in the leaf-width direction (Folkers *et al.,* 2002; Kim *et al.,* 2002). The overexpression of *ROT3* in whole plant organs accelerates elongation of leaves and of floral organs derived from leaves, without affecting their width.

The emerging picture is that cell division is modulated at the organ level probably by signals that define the overall mass of the organ. One gene that seems to function allowing the leaf cells to arrest when needed is *CINCINNATA* (Nath *et al.,* 2003). The interesting aspect of *CIN* is that it links local cell division control to the much sought after systemic signal in leaf development and seems to be required to activate cell division in flowers (Crawford *et al.,* 2004). A detailed review about cell cycle genes can be seen in this issue (Ramirez-Parra *et al.*).

Cell size and endoreduplication

Plant growth happens via cell expansion, a complex process that has been recently reviewed (Martin *et al.,* 2001). Plants have two mechanisms to increase cell size beyond the regular size checkpoint that triggers cell division in eukaryotic cells (Coelho and Leevers, 2000) that might not be mutally exclusive. One is through the action of the vacuole, a specialized organ that allows cell expansion by increasing the intracellular volume without increasing the volume of cytoplasm. A second mechanism is endoreduplication, involving duplication of the genome without mitosis (Joubes and Chevalier, 2000). The increase in cell size correlates with an increased DNA content or ploidy level (Kondorosi *et al.,* 2000), so cell cycle progression and growth, which are normally coupled, are separated. It is thought that the bigger nuclei of polyploid cells meet the requirements of a higher metabolic activity, rRNA synthesis and transcriptional activity in larger cells. The tissue specific pattern of endoploidy is characteristic of the species; in *Arabidopsis* it varies from 4C to 32C. In epidermal pavement cells of *Arabidopsis* morphometry reveals a direct proportionality between nuclear DNA level and cell size (Galbraith *et al.,* 1991). Endoreduplication does not happen in every cell as a mechanism to increase cell size, for instance *Arabidopsis* guard cells expand but maintain a 2C value (Melaragno *et al.,* 1993). Experiments where *Arabidopsis* replication licensing components have been overexpressed elegantly demonstrate that increased cell DNA content happens only in certain cell types (Castellano *et al.,* 2004). Interestingly, plants overexpressing *CDC6* show DNA content phenotypes but do not differ from wild type in terms of body size, suggesting that a general compensatory mechanism is exerted over the growth of the plant. Concerning flower tissues, *Arabidopsis* petals seem to be formed by cells that are largely 2C and endoreduplication has not been observed but in other members of the family, like cabbage, endoreduplication seems to be a common mechanism of petal cell size control (Kudo and Kimura, 2002).

Several mutations affecting cell wall formation in a general way have pleiotropic effects that suggest cell expansion is important to reach fully functional organs. For instance mutations like *ECTOPIC LIGNIFICATION 1* (*ELI1*) where cell expansion and lignification are impaired show pleiotropic phenotypes including decreased floral organ size (Caño-Delgado *et al.,* 2000). The mutant *FRAGILE FIBERS 2* (*FRA2*) is affected in cellulose fiber length and width results in a pleiotropic phenotype consisting of shorter but wider leaves and flowers (Burk *et al.,* 2001, Burk and Ye, 2002). These mutations clearly show that the process of cell wall deposition and formation plays an important role achieving the normal size and proportions of all plant organs. The expansins control cell wall loosening that is important for cell expansion (Cosgrove, 2000). Downregulation of an α-expansin in *Petunia hybrida* preferentially expressed in the petal limb causes a strong reduction in petal limb development whereas the floral tube

formed by the fused part of the petals where the gene is not highly expressed remains normal in the transgenic plants (Zenoni *et al.,* 2004). The phenotypic effects i.e. reduced expansion of the petal limbs (that are called lips in *Antirrhinum*) seems to be due to a decrease in cell expansion in both adaxial and abaxial epidermis. The results of Zenoni *et al.,* suggest that differential growth might be achieved by specific gene expression pattern that give specificity of action to some genes, in this case involved in cell expansion. But it also illustrates that differential gene expression might be the cause of developmental compartments in floral organ development.

Conclusions

Most of the mendelian genes described in this review affect floral organ size together with other aspects of plant development, but the genetic analysis of some mutations and QTL in different plants show that in dicots, some of the regulatory networks controlling organ size might be specific for the flower. It will be interesting to see if the modularity is due to gene redundancy or to genes that are expressed only in the floral context. Some floral specific executors have been found for auxins (*ARG* and *ANT*) or gibberellins (*AP3, PI* and *AG*). We consider these important contributions because like other plant growth regulators they play general roles in development, but the important question is in the specific translation of their function in different tissues.

Nevertheless, new biological pathways will probably arise that are specific for the flower. A conceptual framework about the way cell division, expansion and growth direction are controlled to achieve organ growth and asymmetry has been laid down recently (Coen *et al.,* 2004; Rolland-Lagan *et al.,* 2003). It puts two important aspects in perspective, first the fact that organs might have specific compartments and second the existence of systemic signals that control rate of division, expansion and maybe more important for organ shape, the direction of these two events. Both in animals and plants there is a large body of evidence about this systemic signalling and we believe that one challenge in the future will be to identify mutations in that pathway that shed some light on this complex process.

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