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The Role of the REPLUMLESS Homeodomain Protein in Patterning the *Arabidopsis* Fruit

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Summary

The outside of the *Arabidopsis thaliana* fruit consists of three principal tissues: the valves or seedpod walls, the replum or central ridge between the valves, and the valve margins where the valves separate from the replum to disperse the seeds. Previous studies have shown that valve margin formation is specified by the SHATTERPROOF MADS-box transcription factors [1] and that valve development is controlled by the FRUITFULL MADS-box transcription factor [2]. FRUITFULL negatively regulates SHATTERPROOF to prevent the valves from adopting a valve margin cell fate [3]. Here we identify a gene called *REPLUMLESS* that is required for replum development. *REPLUMLESS* encodes a homeodomain protein that prevents replum cells from adopting a valve margin cell fate by negatively regulating expression of the *SHATTERPROOF* genes. Both REPLUMLESS and FRUITFULL are required to limit *SHATTERPROOF* expression to a narrow stripe of cells so that the valve margin differentiates precisely at the valve/replum boundary.

Results and Discussion

Arabidopsis fruit (Figure 1A) consist of a seedpod that protects the seeds as they mature and disperses them by opening at maturity. The valves are joined to the replum [4, 5] at the valve margins (Figures 1A–1C), and at maturity, separation of valve margin cells allows the valves to detach from the replum, resulting in the dispersal of the seeds [6, 7]. Seed dispersal requires the SHATTERPROOF (SHP1 and SHP2) MADS-box transcription factors, which act redundantly to specify the valve margin [1]. The FRUITFULL (FUL) MADS-box transcription factor plays an important role in valve cell identity since *ful* valve cells fail to differentiate and expand [2] (Figures 2A and 2B). FUL acts in part by repressing expression of the *SHP* genes (Figure 3G), thus preventing valve cells from adopting a valve margin fate [3]. Thus far, genes controlling replum development have yet to be identified.

Because the wild-type replum (Figure 1A) is difficult to observe without the aid of a microscope, we took advantage of the enlarged, twisted replum of *ful* mutant fruit (Figures 2A–2C) [2, 8] to screen for novel mutants affecting replum development (see Supplemental Experimental Procedures at http://www.current-biology.com/cgi/content/full/13/18/1630/DC1). We identified a double mutant, *replumless* (*rpl*) *ful*, that affects the architecture of the plant (see Supplemental Data) and appears to lack the replum (Figure 2D). In *rpl ful* mutant fruit, cells that would normally form the replum instead remain small (Figure 2E), similar to the *ful* mutant valve cells that surround the replum. Thus, the entire circumference of the *rpl-1 ful* fruit is covered with small cells without any obvious delineation between valve, valve margin, or replum regions (Figure 2D). Since the small modified valve cells in *ful* mutants are known to have adopted a partial valve margin identity [3], their similarity to cells in the *rpl-1 ful* replum region suggests that replum cells in *rpl-1 ful* may also have taken on a valve margin cell fate. A cross-section shows that the disruption in the *rpl-1 ful* replum development is limited to the outer replum cell layers and that the inner parts of the replum including the vascular bundles are present (Figures 2C and 2F).

To determine if the replum defect is also present in the *rpl-1* single mutant, *rpl-1 ful* double mutants were crossed to wild-type to remove the *ful* mutation and additional single mutant alleles were isolated subsequently. The overall fruit morphology of the *rpl-1* single mutant is similar to wild-type, except that the mutant fruit are about half as long as wild-type (Figure 1D). Close inspection of the *rpl* replum region (Figures 1E, 1F, and 1I–1L) reveals that replum cells have been replaced by narrow files of cells that resemble cells found in the valve margin (Figures 1B and 1C). In the most extreme cases, such as in *rpl-3* fruit (Figures 1K and 1L), the number of cell files is reduced and the valves appear to have encroached into the replum region.

To determine whether *rpl* replum cells have adopted a valve margin identity, we examined the expression patterns of GT140 [1, 3, 9] and SHP2::GUS [10], two molecular markers known to be expressed in stripes at the valve margin. In contrast to wild-type, both GT140 and *SHP2::GUS* are ectopically expressed in the replum region of the *rpl* mutant (Figures 3A, 3B, 3E, and 3F). These data demonstrate that the *RPL* gene is required to prevent the ectopic expression of valve margin markers in the replum and are consistent with the appearance of valve margin-like cells in the replum region of *rpl* mutants.

The initial expression domain of *SHP2::GUS* in both wild-type and *rpl* mutants is relatively broad (stages 8–11) [11], encompassing cells that will later form the valve margin and replum (Figures 3C and 3D). However, shortly before fertilization when the valves, valve margins, and replum are just beginning to become morphologically distinct (by stage 12) [12], SHP2*::GUS* expression becomes restricted to the valve margins (Figure 3E). In contrast, *SHP2::GUS* expression continues to be detected in the replum region of *rpl-1* mutants (Figure 3F), where it remains late into fruit development (stage

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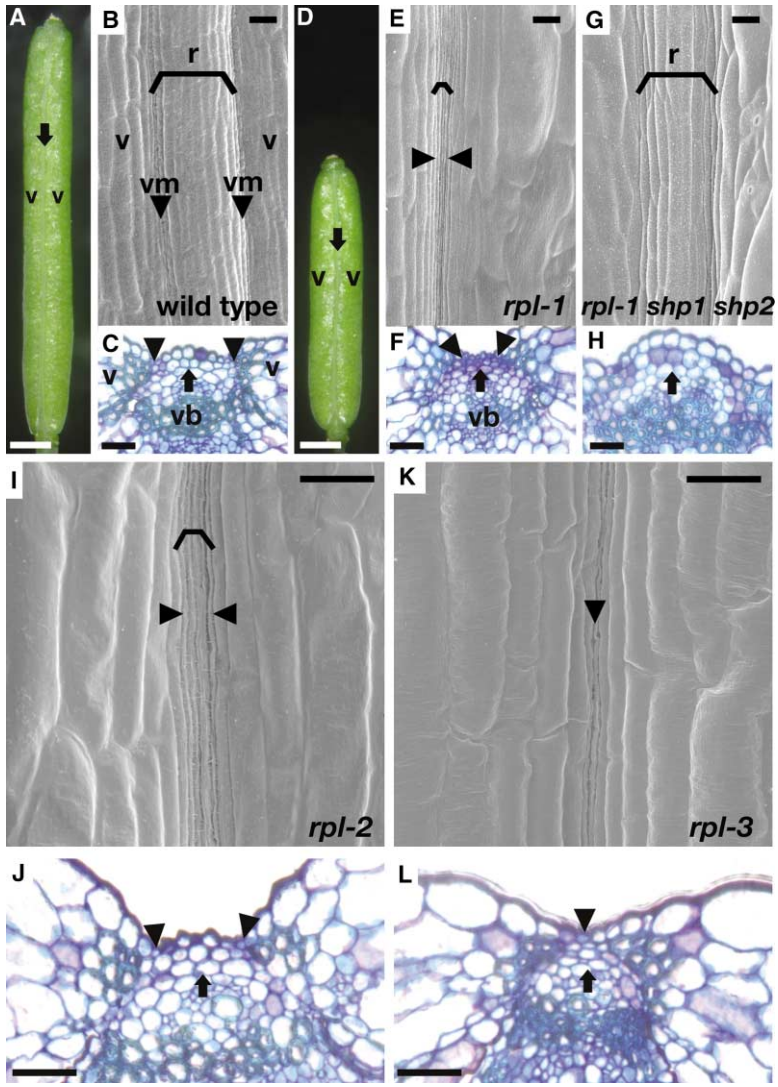

Figure 1. *RPL* Is Required for Replum Formation

(A–C) In wild-type (Ler) stage 17 fruit, the replum (arrow in A and C, r bracketed in B) is the ridge of cells between the valves (v). The replum was classically defined as the structure that remains attached to the plant after the valves have fallen from the fruit at maturity and includes the septum [4]. Here we use the more recent definition in which the replum is defined as the outer or abaxial region that does not include the internal septum [5]. The valve margins (vm arrowheads in B and C), where the valves join the replum, are composed of narrow cells.

(D–F) In the *rpl-1* fruit, the replum region (arrow in D and F, bracketed in E) contains narrow cells that are similar to valve margin cells. Only the outer cell layers are affected and the inner vascular bundle (vb) is present (F).

(G and H) In *rpl-1 shp1 shp2* triple mutant fruit, replum development is restored (r bracket in G, arrow in H).

(I and J) The *rpl-2* phenotype is comparable to *rpl-1* since small cells similar to valve margin cells replace the replum.

(K and L) The *rpl-3* allele has a more severe phenotype with a reduced number of cell files in the replum region (arrowhead). Defects in the fusion of the septum are also present in *rpl-2* and are prominent in *rpl-3* mutant plants.

Panels (B), (E), (G), (I), and (K) are scanning electron micrographs (SEMs). Panels (C), (F), (H), (J), and (L) are cross-sections of the replum region. The scale bars in (A) and (D) equal 1 mm and the scale bars in (B), (C), and (E)–(L) equal 20 μm.

17, data not shown). These data indicate that *RPL* is initially required to prevent the ectopic expression of the *SHP2::GUS* marker during stage 12. Previous studies have shown that *SHP2* is ectopically expressed throughout the valves of *ful* mutants, indicating that FUL negatively regulates *SHP* expression (Figure 3G) [3]. We have found that the expression domain of *SHP2::GUS* surrounds the fruit in *rpl-1 ful-5* double mutants (Figure 3H), apparently accounting for the uniformly small valve margin-like cells encircling the double mutant fruit (Figures 2D–2F).

Since the *SHP* genes specify valve margin development and are ectopically expressed in the *rpl* mutant replum, we tested whether this ectopic *SHP* expression causes the replum cells to take on valve margin cell fates. We removed SHP activity by constructing the *rpl-1 shp1 shp2* triple mutant and found that replum formation was restored, indicating that the ectopic expression of the *SHP* genes is largely responsible for the loss of replum development in *rpl* mutants (Figures 1G and 1H). Replum differentiation was similarly restored in *rpl-1 ful shp1 shp2* quadruple mutants (Figures 2G–2I, see Supplemental Figure S2 at http://www.current-biology.com/cgi/content/full/13/18/1630/DC1). Taken together, these data indicate that RPL is not directly required for replum formation, but is instead required to prevent the expression of *SHP* in replum cells. The development of replum cells in *rpl shp1 shp2* mutants further suggests that an underlying pattern for replum development has already been established and that the subsequent role for RPL is to prevent these cells from adopting a valve margin cell fate.

The wild-type valve margin consists of a lignified layer (Figure 3M), which is proposed to provide tensions that contribute to pod opening [13], and a separation layer (Figure 3I), where separation of the cells leads to detachment of the valves from the replum [14]. Since the *SHP* genes control the formation of both the lignified and separation layers and because the *rpl* replum cells have adopted valve margin characteristics, we tested whether these cells resemble separation layer cells or lignified layer cells instead of replum cells. Separation layer cells stain

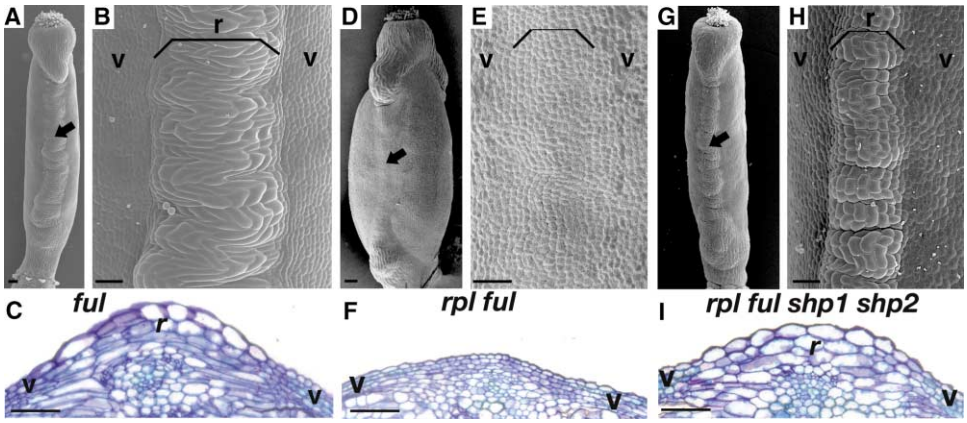

Figure 2. The Enlarged Replum of *ful* Mutants Fails to Develop in *rpl-1 ful* Double Mutants but Is Partially Restored in *rpl-1 ful shp1 shp2* Quadruple Mutants

(A and B) In *ful-1* fruit at stage 17 (A and close-up of the replum in B), the replum (arrow in A, r bracketed in B) is enlarged and twisted (compare to Figures 1A and 1B).

(D and E) These large twisted replum cells are absent in the *rpl-1 ful-1* fruit (D and close-up in E). Occasionally *rpl ful* fruit were observed with fully developed repla often only on one side of the fruit, indicating that the phenotype is incompletely expressed.

(C and F) The *ful-1* and the *ful-5* alleles are both strong mutant alleles and the fruit phenotypes are nearly identical. A cross-section of the *ful-5* replum (C) shows that both the diameter of the cells and the overall width of the replum are enlarged. A cross-section of the *rpl-1 ful-5* replum (F) shows that the cells are small and similar to the surrounding valve cells (v).

(G–I) Replum development is partially restored in *rpl-1 ful-5 shp1 shp2* quadruple mutants. A range of replum development was seen in *rpl-1 ful-5 shp1 shp2* from fairly enlarged repla to just enlarged cells that are clearly different from those of *rpl-1 ful-5*, but not as wavy as *ful-5 shp1 shp2* repla. Even the largest repla of *rpl-1 ful-5 shp1 shp2* were not as large as those of *ful-5 shp1 shp2*, but replum development was most similar to *ful-5 shp1 shp2* near the base of the fruit. Replum development in *ful-5 shp1 shp2* fruit is similar to that in *ful* fruit (see Supplemental Figure S2).

(A), (B), (D), (E), (G), and (H) are SEMs. (C), (F), and (I) are cross-sections of the replum region. Scale bars in (A), (D), and (G) are 100 μm and scale bars in (B), (C), (E), (F), (H), and (I) are 50 μm.

blue when stained with safranin O and alcian blue [14, 15] (Figure 3I), while lignified layer cells stain pink when stained with phloroglucinol [1] (Figure 3M). In most *rpl-1* fruit, the replum region stains light blue, indicating that it has adopted characteristics of the separation layer (Figure 3J). In the *rpl-1 ful* double mutant fruit, these small blue separation layer cells cover the entire replum region (Figures 3K and 3L). In the more severely affected *rpl* fruit (as often occurs in *rpl-3* fruit), the lignified layer extends across the replum region (Figure 3N). These data suggest that there is a gradation of phenotypes from less extreme (where the replum region takes on characteristics of the separation layer) to more severe (where the lignified layer intrudes into the replum region, and in the most extreme cases the lignified layer connects across the replum).

We next tested whether the loss of normal replum development in *rpl* mutants affects the opening of the fruit, or fruit dehiscence. In the strong *rpl-3* allele, a decrease in fruit dehiscence was observed (*rpl-3*: 20% dehiscent, 50% slightly indehiscent, 29% moderately indehiscent, and 1% severely indehiscent, n = 202) as compared to wild-type (Wassilewskija (WS): 97% dehiscent, 3% slightly indehiscent, n = 201). Only a very slight decrease in fruit dehiscence was observed in the less severe *rpl-1* fruit (*rpl-1*: 84% dehiscent, 16% slightly indehiscent, n = 200) when compared to wild-type fruit (Landsberg erecta (Ler): 95% dehiscent, 5% slightly indehiscent, n = 201). The decrease in fruit dehiscence in *rpl-3* fruit is apparently due to the extension of the lignified layer across the replum region, preventing the valves from separating from one another even after they have detached form the inner replum (Figure 3O). These results suggest that one role for the outer replum is to prevent the valve margin lignified layers from fusing together and inhibiting dehiscence.

We used a map-based approach to clone the *RPL* gene and found that it encodes a putative homeodomain transcription factor (At5g02030) (Figure 4A). We verified that this gene corresponds to *RPL* by rescuing the mutant phenotype with a wild-type copy of the *RPL* gene (compare Figures 4B and 4C with Figures 2D and 2E) and by characterizing two independently isolated insertion mutant alleles (Figures 4A and 1I–1L). Both *rpl-2* and *rpl-3* displayed the same overall phenotypes as *rpl-1*. The *rpl-3* allele exhibited the most severe phenotypes (Figures 1K and 1L), perhaps because it is a stronger allele (Supplemental Figure S3 at http://www.current-biology.com/cgi/content/full/13/18/1630/DC1) or possibly due to the WS accession in which it was isolated.

RPL belongs to the BELL1 family of homeodomain transcription factors (Supplemental Figure S4) [16–18]. Previous studies have shown that BELL1 regulates ovule development in part by negatively regulating *AGAMOUS* [19, 20], a MADS-box gene closely related to the *SHP* genes. Here we show that RPL has a similar function in negatively regulating the *SHP* MADS-box genes during replum development. It will be interesting to determine whether repression of MADS-box transcription factors is a common role for the other members of the BELL1 family.

RPL is expressed in all tissues tested, with the highest

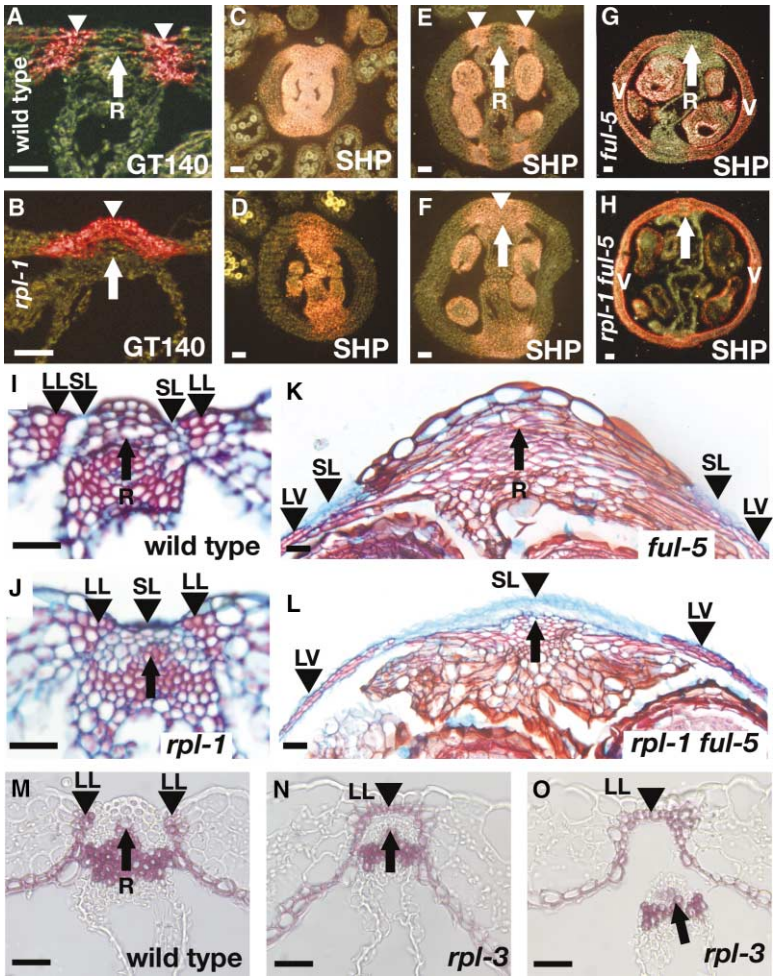

Figure 3. Markers for the Valve Margin Are Expressed in the *rpl-1* Replum Region

Molecular markers for the valve margin were examined in wild-type and *rpl* under dark field optics where the marker expression appears pink and the unstained tissue appears white.

(A and B) The GT140 molecular marker, which is expressed in the wild-type (A) valve margin (arrowheads), is ectopically expressed in the *rpl-1* (B) replum (arrow) as shown in cross-sections of the replum regions of stage 17 fruit.

(C–H) The *SHP2::GUS* reporter for *SHP2* expression (noted as *SHP*) is initially expressed broadly in the wild-type developing ovary (C) (cross-section of stage 10) but is limited by the end of stage 12 to the valve margin and ovules (E). *SHP2::GUS* is also initially expressed broadly in *rpl-1* ovaries (D) (stage 10), but at stage 12, *SHP2::GUS* is not limited to the valve margin and expression continues in the replum (F). In a cross-section of the middle of *ful-5* fruit at stage 17, *SHP2::GUS* is expressed throughout the valves (v) (G). In *rpl-1 ful-5* fruit, *SHP2::GUS* surrounds the fruit including the replum (arrow) and valves (v) (H).

(I–L) Cross-sections of replum regions were stained with safranin O and alcian blue at late stage 17 or early stage 18. In wild-type fruit (I), the separation layer of the valve margin (arrowhead SL) stains blue while the replum stains red (arrow) and the lignified layer of the valve margin (arrowhead LL) stains pink. Separation of the valve from the replum is already beginning to occur on the left. In *rpl-1* (J), the blue staining typical of valve margin separation layer cells covers the replum. In *ful-5* (K), the valves stain pink, indicating that they are lignified (arrowhead LV). As in wild-type, the large *ful-5* replum stains red and the separation layer stains blue. In *rpl-1 ful-5* (L), the replum region stains blue similar to the separation layer, suggesting a replacement of replum cells by valve margin cells.

(M–O) Cross-sections of the replum region were stained with phloroglucinol to detect lignification (pink). In wild-type (WS) late stage 17 fruit, the lignified layer can be detected at the valve margins (arrowhead LL) (M). In some parts of the *rpl-3* stage late 17 fruit, the lignified layer extends across the replum (arrowhead LL) (N). In some *rpl-3* stage 18 fruit, the inner replum and septum have separated from the valves, but the valves remain attached through the lignified layer (arrowhead LL) (O).

Scale bars are 25 μm.

levels detected in stems, inflorescences with flower buds, and open flowers (Figure 4D). In situ hybridization of wild-type flowers showed that *RPL* is expressed in the replum early in flower development (stages 7 and 8) (Figure 4G). We also generated transgenic plants containing a GUS reporter under the control of the putative *RPL* regulatory region and found that, in agreement with the in situ data, *RPL::RPL-GUS* was expressed in the replum (Figure 4H). *RPL::RPL-GUS* expression in the replum was strong at stage 12 (Figures 4F and 4I), which coincides with the time RPL is required to repress *SHP* expression in the replum. *RPL::RPL-GUS* expression was also observed in the style, in the stem and pedicels, in the sepal vasculature (Figure 4E), and in the inflorescence meristem (data not shown).

In conclusion, we propose that the RPL homeodomain protein acts as a transcription factor in the replum to negatively regulate *SHP* gene expression, thereby preventing the replum from adopting a valve margin cell fate (Figure 4J). Similarly, FUL acts in the valves to repress *SHP* expression and to prevent valve cells from adopting a valve margin fate [3]. Together, negative regulation by RPL and FUL defines the narrow stripes of *SHP* gene expression, restricting valve margin development to the valve/replum boundary.

Although the *Arabidopsis* fruit structure is typical of several thousand species of Brassicaceae, including oilseed crops such as canola (*Brassica napus*) [21], the replum morphology varies considerably [22]. For example, the replum of *Allairia petiolata* (M. Bieb) of Cavara et Grande fruit is very large and protrudes from the fruit in a manner that is reminiscent of the *ful* mutant replum. In contrast, *Brassica napus* fruit form a suture with no external replum where the valve margins come together in a V shape [14], which is reminiscent of the *rpl-3* fruit. Now that we have begun to understand the genetic interactions that pattern the *Arabidopsis* fruit, it will be interesting to determine the extent to which this patterning mechanism is conserved in different plant species and if differences in the expression and function of *RPL, SHP,* and *FUL* contribute to the differences in fruit morphology seen in diverse plant species.

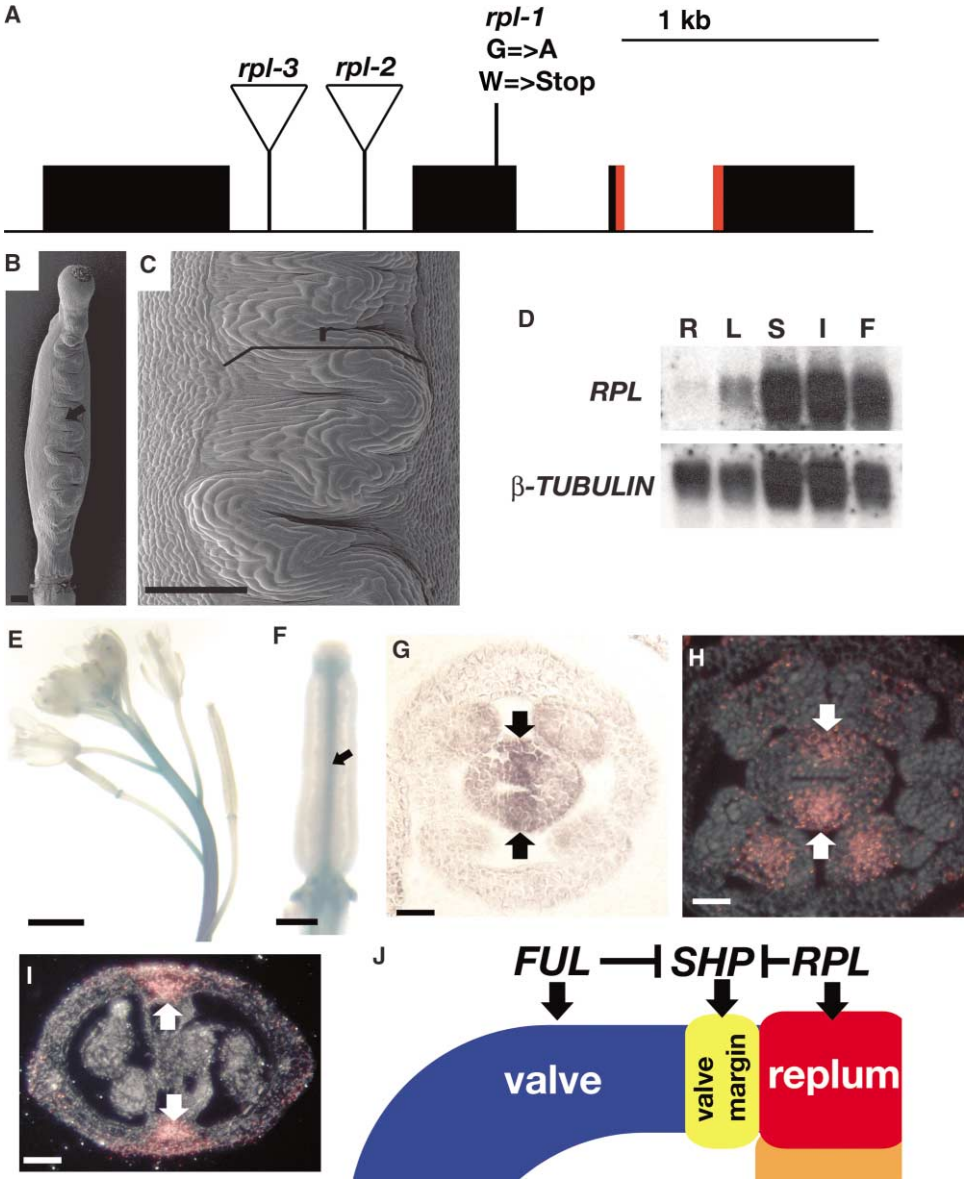

Figure 4. *RPL* Encodes a Homeodomain Protein, and *RPL::RPL-GUS* Is Expressed in the Replum

(A) *RPL* encodes a homeodomain protein (At5g02030) of four exons (boxes). The locations of the homeodomain (red) and the mutations in each allele are shown. The T-DNA sizes are not shown to scale.

(B and C) A wild-type copy of the *RPL* gene (pAR34B) completely rescues replum (arrow in B and r bracket in C) formation in *rpl-1 ful* plants as seen in SEMs.

(D) A blot of polyadenylated RNA shows that *RPL* is expressed at very low levels in roots (R), medium levels in rosette leaves (L), and higher levels in stems (S), inflorescences including flowers to stage 12 (I), and flowers stages 14 to 16 (F). β-*TUBULIN* was used to probe the same blot as a loading control.

(E–I) The *RPL::RPL-GUS* reporter (blue expression) is expressed strongly in the stem and pedicels (E) as well as in the replum (arrow) (F [stage 12] and cross-sections in H [stage 7] and I [stage 12] shown in dark field where expression is pink). (G) In situ hybridization with a *RPL*-specific probe (pAR45) shows that *RPL* is expressed in the replum of developing ovaries (stage 7).

(J) A model showing how RPL and FUL act together in fruit development to limit *SHP* expression to narrow stripes at the border between the valves and the replum.

Scale bars equal 200 μm in (B), (C), (F), and (I); 25 μm in (G) and (H); and 2 mm in (E).

Supplemental Data

Supplemental Data, including information on the plant phenotype, the BELL1 family of proteins, the *RPL* alleles, and methodological details, can be found at http://www.current-biology.com/cgi/content/full/13/18/1630/DC1.

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Note Added in Proof

While this manuscript was under review, two papers were published about the internode elongation defect seen in *rpl* mutants. These papers refer to the gene as PENNYWISE (Smith, H.M.S., and Hake, S. (2003). The interaction of two homeobox genes, BREVIDPEDICELLUS and PENNYWISE, regulates internode patterning in the *Arabidopsis* inflorescence. Plant Cell *15*, 1717–1727) and as BELLRINGER (Byrne, M.Y., Groover, A.T., Fontana, J.R., and Martienssen, R.A. (2003). Phyllotactic pattern and stem cell fate are determined by the *Arabidopsis* homeobox gene BELLRINGER. Development *130*, 3941–3950).