

Figure S1. Phenotypes of *atbmi1a/b* and *atbmi1a/b/c* mutants. (A) WT seedling at 10 DAG. (B) *atbmi1a/b* mutants at 10 DAG. (C) *atbmi1a/b/c* mutants at 10 DAG. Bars, 2 mm.

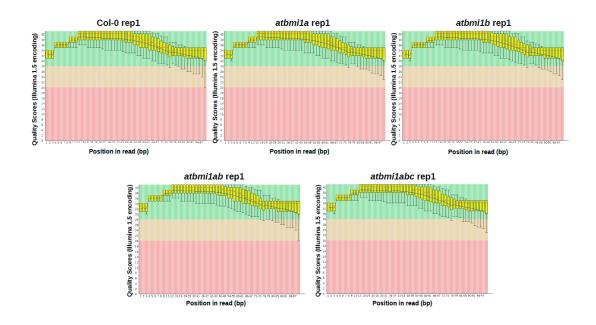


Figure S2. Boxplots representing the read quality scores (Illumina 1.5 encoding) per base for the first replicate of all samples. The quality scores for each base in the reads remained within the green area indicating a high sequencing quality. The common decrease in quality at the end of the reads is observed. Nevertheless, the quality never enters the problematic red area.

SAMPLE	NUMBER OF READS	CONCURRENT PAIR ALIGNMENT RATE
WT Col0 rep1	14578745	96.5%
WT Col0 rep2	16253159	96.6%
atbmi1 a rep1	14982986	94.5%
atbmi1 a rep2	18324516	96.1%
atbmi1 b rep1	15714678	95.8%
atbmi1 b rep2	18412180	95.3%
atbmi1 ab rep1	14885215	94.9%
atbmi1 ab rep2	14547885	95.1%
atbmi1 abc rep1	14832480	94.5%
atbmi1 abc rep2	12349849	94.9%

Table S1. Number of reads and concurrent pair alignment rate per sequencing sample. On average the number of reads per sample is approximately 15 million and the average concurrent pair alignment rate is greater than 95.%. This indicates a high read sequencing quality and the lack of sample contamination.

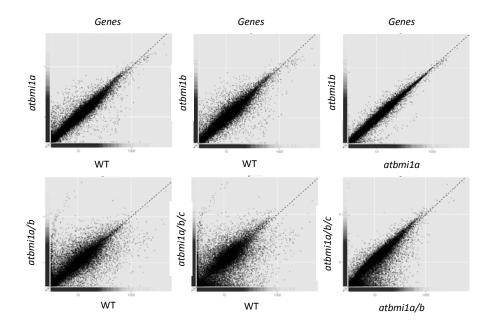


Figure S3. Correlation among differentially expressed genes in WT and the different genotypes. Scatter plots comparing gene expression levels in the different mutants against WT, single *atbmi1a* against *atbmi1b*, and *atbmi1a/b* against *atbmi1a/b/c*.

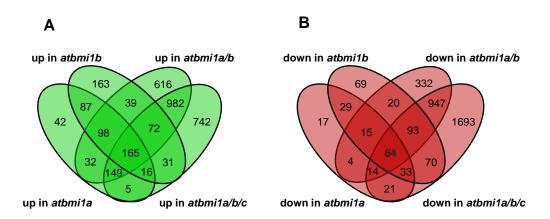
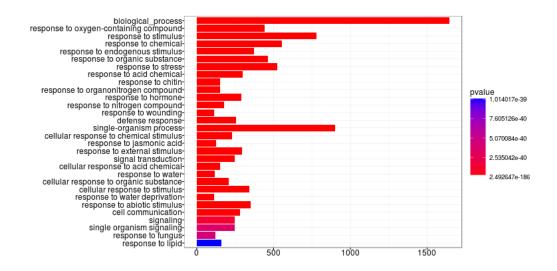


Figure S4. Altered gene expression in *atbmi1* mutants. (A) Venn diagram showing the number of genes up- and (B) downregulated that overlap among the different genotypes.

GO of genes upregulated in atbmi1a/b



В

GO of genes downregulated in atbmi1a/b

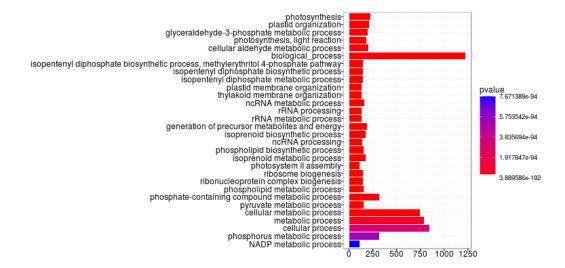
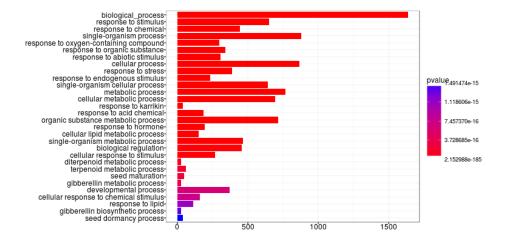


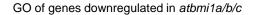
Figure S5. Gene ontology (GO) enrichment analysis of up- and downregulated genes in *atbmi1a/b* mutants. Distribution of enriched GO terms into the different "biological process" categories as defined by TAIR. p-values are indicated.

Α

GO of genes upregulated in atbmi1a/b/c



В



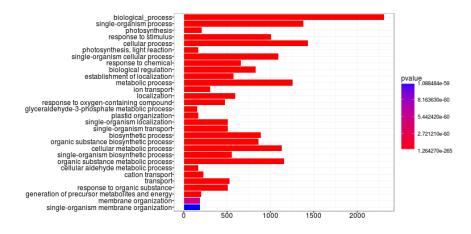
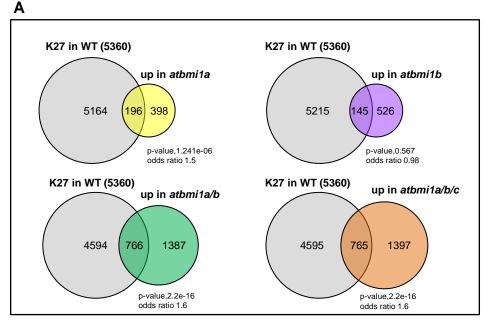


Figure S6. Gene ontology (GO) enrichment analysis of up- and downregulated genes in *atbmi1a/b/c* mutants. Distribution of enriched GO terms into the different "biological process" categories as defined by TAIR. p-values are indicated.

Α



В

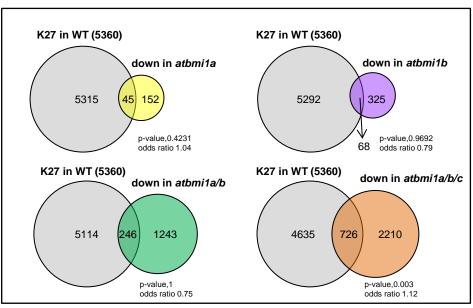


Figure S7. Putative AtBMI1direct target genes. (A) Venn diagrams showing the number of genes that were upregulated (up) in the different mutants and H3K27me3 marked (K27) in WT seedlings of the same age. All these overlaps are significant with p-values lower than 1.2 x10⁻⁶ and odds ratios greater 1.5 according to Fisher's Exact test except in the case of the *atbmi1b* mutant, which is probably because it is a knock-down mutant. **(B)** Venn diagrams showing the number of genes that were downregulated (down) in the different mutants and H3K27me3 marked (K27) in WT seedlings of the same age. All these overlaps are non-significant with p-values higher than 0.4231 and odds ratios lower than 1.044 according to Fisher's Exact test except in the case of the *atbmi1abc* mutant, which is probably because the developmental stage of the mutant.



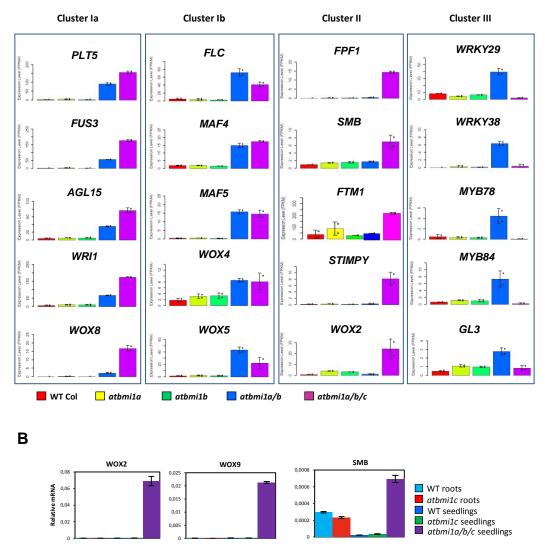


Figure S8. Genes differentially expressed in *atbmi1a/b* and *atbmi1a/b/c.* (A) Expression levels of several genes from the different clusters in WT seedlings and the different mutants. (B) qRT-PCR analysis of *WOX2*, *WOX9* and *SMB* expression levels y whole seedlings and roots of WT, *atbmi1c* and *atbmi1a/b/c* mutants. Quantifications are relative to *ACTIN* levels. Error bars of three independent measurements are indicated.

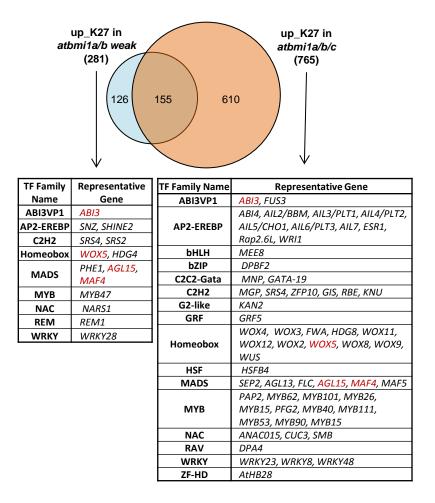
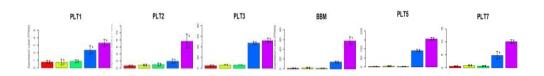
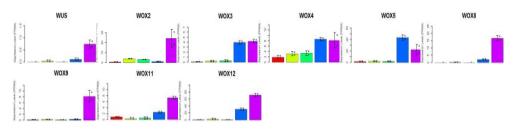


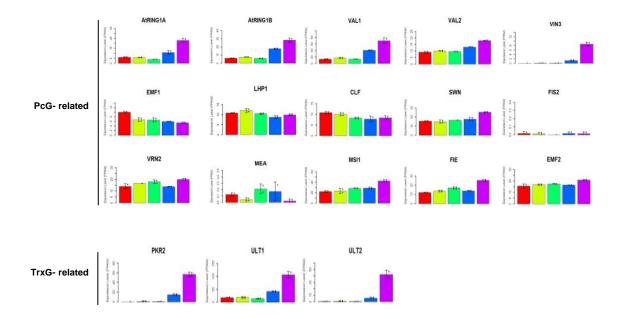
Figure S9. Different gene expression patterns of *atbmi1a/b* weak and *atbmi1a/b/c* **mutants.** Venn diagram showing overlapping between the genes up_K27 in *atbmi1a/b* weak and *atbmi1a/b/c* mutants. The overlap is significant with a p-value lower than 2.2x10⁻¹⁶ and an odds ratio greater than 17 according to Fisher's Exact test. Some representative transcription factors (TFs) in each dataset are indicated. TFs found in the two data sets are highlighter in red.



WOX gene family

PLT gene family





CHROMATIN Factors

Figure S10. Expression levels of different important developmental genes in WT and *atbmi1a/b/c* mutants. Transcript levels of genes from *PLT* and *WOX* gene families and chromatin related factors belonging to the PcG and TrxG families.

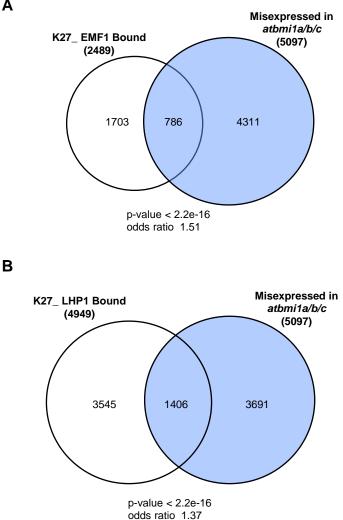


Figure S11. AtBMI1, EMF1 and LHP1 functional relationship. (A) Clustering analysis of genes misexpressed (up and downregulated) in atbmi1a/b/c and H3K27me3 marked genes bound by EMF1. (B) Clustering analysis of genes misexpressed (up and downregulated) in atbmi1a/b/c and H3K27me3 marked genes bound by LHP1. These overlaps are significant (p-values and Fisher's Exact test results are indicated).

Α

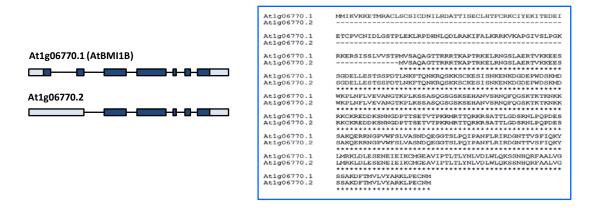


Figure S12. Schematic representation of *AtBMI1B* (*At1g06770*) splice variants (left) and predicted protein sequence comparison (right). Light boxes indicate untranslated regions, blue boxes exons, and black lines introns.