Figure S1.—Screening method of an AFLP marker tightly linked to the FT2 locus using RHL derived from RIL6-8. (A) Graphical genotype of RHL6-8 and NILs are shown on the left side of the figure. The white, black and gray chromosomal segments represent the Misuzudaizu, the Moshidou Gong 503 and heterozygous alleles, respectively. The first screening of AFLP markers was performed using NILs6-8 (No 1 and 2). Polymorphic markers detected between NILs were reanalyzed using subfamilies selected from the progenies of RHL6-8 (No 3-8). Five AFLP fragments tightly linked to the FT2 locus were highly reproducible and were used in subsequent fine mapping experiments. Representative segregation patterns of each fragment among the NILs and RIL population using one AFLP marker, E37M31, are shown in the box and in B, respectively. Arrowhead indicates polymorphic band detected between NILs and asterisks indicate the polymorphic markers detected between Misuzudaizu and Moshidou Gong 503 originating from other genetic loci.
Figure S2.—The physical contig covering the FT2 locus. SCAR markers originating from AFLP fragments identified 10 BAC clones in this study. Left and right sequences of each BAC clone were deposited in DDBJ under accession numbers AB554201-AB554220. Positional relationships among the DNA markers using fine mapping experiments (A) and the physical contig (B) are displayed. Five BAC clones, indicated by bold lines, were subject to shotgun sequence analysis and their nucleotide sequences deposited in DDBJ under accession numbers AP011822 (WBb35C13), AP011811 (MiB300H01), AP011821 (WBb225N14), AP011813 (MiB319A04) and AP011810 (MiB039C03), respectively.
Figure S3.—Diagnostic derived cleaved amplified polymorphic sequences (dCAPs) marker detection of a premature stop codon in GmGIa. (A) The position of the stop codon mutation, the primer sequences and the specific DraI site amplified with the dCAPs primers are displayed. Detailed primer sequences and experimental conditions of this dCAPS marker are shown in the Materials and methods. (B) Representative NILs mentioned in this study were analyzed (No. 1-8). PCR products, without or following DraI restriction enzyme digestion, are compared. Arrows indicate the specific band originating from e2 allele (GmGIa).
Figure S4.—Screening and characterization of a GmGIa mutant line. (A) A mutant library comprising of approximately thirty one thousand of M2 plants were examined. Formation of a heteroduplex and following CEL I digestion of the PCR fragment, amplified from a mixed template of the mutant and wild type DNA (M/C), exhibits a specific truncated DNA band because of mismatch sequences as determined by agarose gel electrophoresis. On the other hand, a non-mixed template (M/M) does not show the specific band. Mutation sites detected in each mutant was validated by direct sequencing of the PCR fragment. (B) One mutant line, 244-A-7, harboring a single nucleotide deletion in the 10th exon, and causing a premature stop codon, was used in the phenotypic evaluation (see text).
Tables S1-S7 are available for download as an Excel file at http://www.genetics.org/cgi/content/full/genetics.110.125062/DC1.

Table S1: Data for standard deviation in RILs
Table S2: Raw data for flowering time in RILs
Table S3: Phenotypic variation in parental lines and RHL6-8
Table S4: Raw data for fine mapping experiments
Table S5: Frequency of flowering time classified with the line and QTL genotype
Table S6: Average values for fine mapping and NIL evaluation
Table S7: Raw data for progeny test