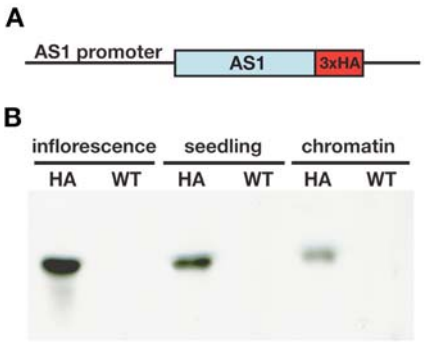
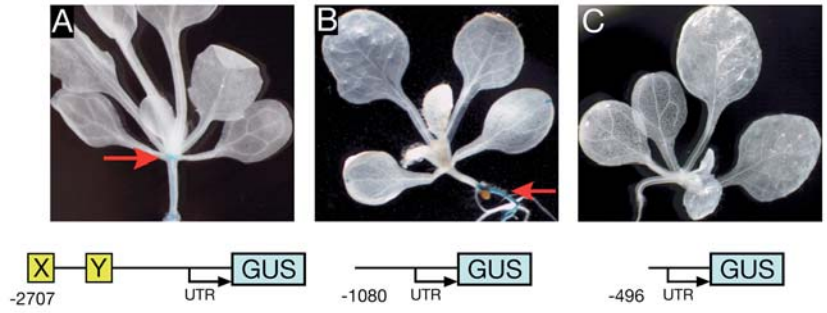
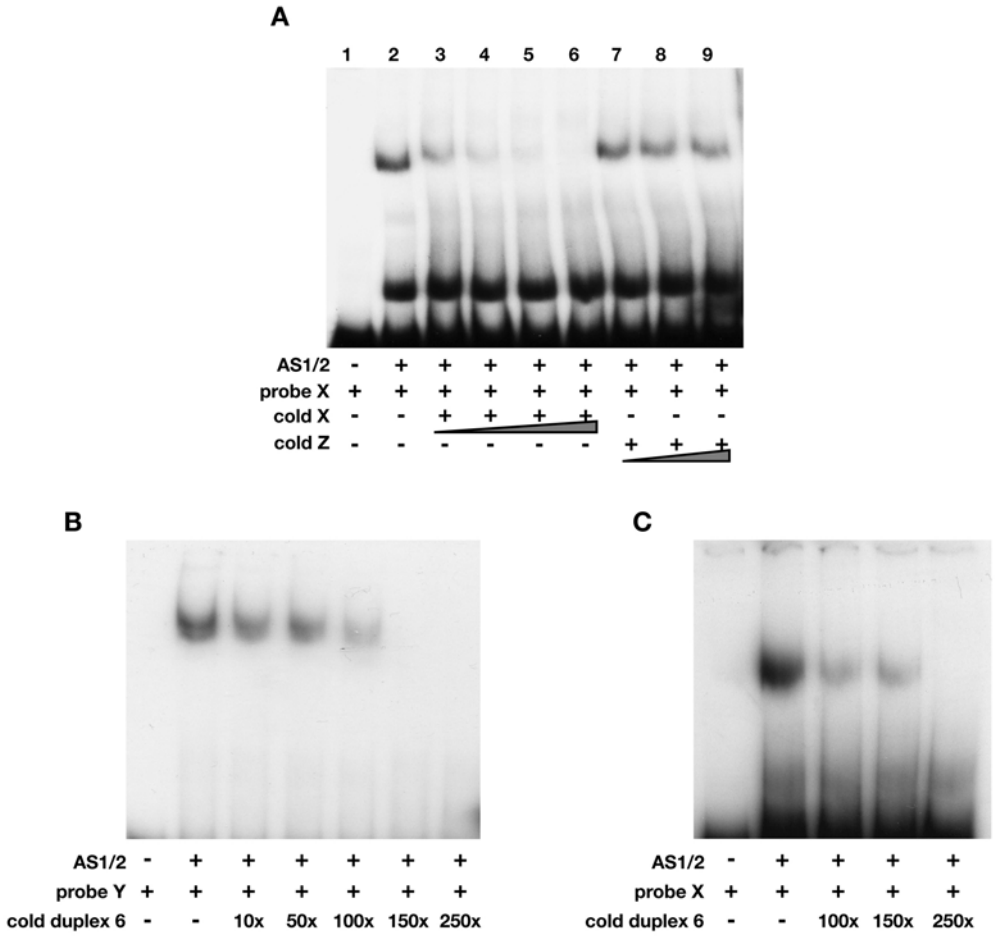


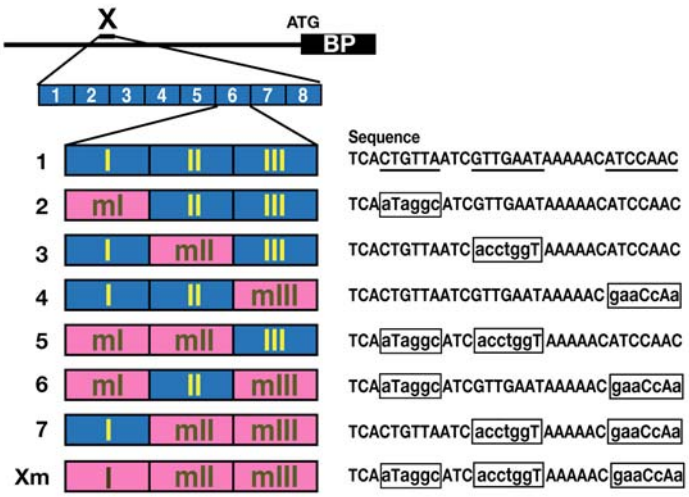
Supplemental Figure 1. Monoclonal HA antibodies specifically recognize the AS1-HA fusion protein. (A) Diagram of the *AS1*$_{pro}$*>AS1-HA* insert. The AS1 protein with a C-terminal 3x HA-epitope tag under control of *AS1* regulatory sequences was transformed into *as1-1/+* plants. Phenotypically normal T2 lines homozygous for *as1-1* as well as the transgene were selected for further analysis. (B) Total soluble protein prepared from wild type (WT) and *AS1*$_{pro}$*>AS1-HA* inflorescences, 2-week-old seedlings or chromatin extracts was analyzed by western. Incubation with the primary monoclonal HA antibody 12CA5 (1:5000 dilution) followed by horseradish-peroxidase anti-mouse IgG secondary antibody (1:2000 dilution), revealed that HA-antibodies specifically recognize the AS1-HA fusion protein.



Supplemental Figure 2. Expression analysis of additional *BP* promoter deletion constructs. (A) The AS1-complex binding fragments X and Y identified by ChIP are located between nucleotides 2707-2522 and 2038-1788 upstream of the BP translation initiation site, respectively. When fused to the GUS reporter, a *BP* promoter fragment that includes both AS1-complex binding sites but no sequences 5' to fragment X drives expression in the SAM, hypocotyl and root, but not in developing leaves. This promoter region thus recapitulates the 3.5 kb *BP* promoter GUS reporter (Figure 3A), as well as the described *BP* mRNA expression pattern in wild type, indicating it contains the regulatory elements sufficient for *BP* expression in the SAM and for the stable silencing of *BP* in leaves. (B) A 1080 bp region upstream of the BP start codon drives expression only in the root, indicating that enhancer elements required for *BP* expression in aerial parts of the plant, SAM and leaves, are located upstream. (C) Upon further deletion of nucleotides 1080-496 upstream of the BP translation start site, all promoter activity is lost as no GUS reporter activity is detectable.



Supplemental Figure 3. Specific binding of AS1-AS2 heterodimers to *BP* promoter fragments X and Y. (A) When co-translated, AS1 and AS2 bind to fragment X in the *BP* promoter (lane 2). Competition assays confirm that this binding is specific. Addition of unlabeled fragment X to the binding assay at 50, 100, 150, or 250 fold molar excess competes effectively with AS1-AS2 binding to probe X (lanes 3-6). In contrast, increasing amounts (50x, 100x, 250x) of unlabeled fragment Z, which based on ChIP does not interact with the AS1-complex *in vivo*, has no effect on AS1-AS2 binding to X (lanes 7-9). (B, C) Competition assays reveal conserved AS1-AS2 binding motifs in *BP* promoter fragments X and Y. Unlabeled duplex 6 was added to the binding assay at increasing molar excess as indicated below each lane. This shows that duplex 6 competes with binding of AS1-AS2 to both fragments Y (B) and X (C). However, a higher molar excess of duplex 6 is required to fully compete binding of AS1-AS2 to fragment X, suggesting that the AS1-AS2 complex binds with higher affinity to this site.



Supplemental Figure 4. Sequences of duplex 6 variants used in competition assays. Duplex 6 is essential and sufficient for AS1-AS2 binding. Its sequence, shown on the first line, includes the c-Myb consensus binding site CNGTTR in region I, and the palindromic sequence GTTGa/gAT in regions II and III (underlined). The sequences of the duplex 6 variants with mutations in individual or combinations of these regions are aligned below.