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Genetic mapping of a chromatin-level modifier

In many organisms, gene activity can be regulated by packaging of the DNA into chromatin. In general, loose chromatin structure is associated with transcriptionally active genes, while inactive genes have tighter chromatin structure. This tighter structure may be associated to increased DNA methylation levels. In order to understand this level of regulation, our lab is studying the purple plant (Pl) gene, which regulates synthesis of purple anthocyanin pigments. Pl-Blotched is an allele that shows a variegated, rather than uniformly-purple, phenotype and this phenotype has been correlated with lower levels of Pl mRNA, tighter chromatin structure and altered patterns of DNA methylation. The dominant modifier, Suppressor of plant blotching (Spb), increases expression of Pl-Blotched. Plants carrying both Pl-Blotched and the dominant allele of Spb have a deep purple color, while plants without Spb have a variegated phenotype and are lighter in color. Increased pigment levels in Spb plants are correlated with increases in Pl mRNA and changes in methylation of Pl-Blotched DNA. These observations suggest that Spb modifies Pl-Blotched. By understanding how this regulation occurs, we can better understand chromatin-level regulatory mechanisms. Mapping Spb is the first step to understanding how it modifies Pl-Blotched. This will allow a cloning strategy to be developed, Spb to be cloned, and eventually, analysis of Spb to take place, which will help us understand how it regulates anthocyanin production in maize. To determine the genetic map location of Spb, I analyzed F2-derived F3 plants from two mapping populations in which Spb was segregating. Pigment phenotypes had previously been determined for the F2 plants in these populations. For genotyping, I used a set of simple sequence repeat (SSR) markers that were evenly dispersed across the genome. If Spb is linked to one of these markers, then we expect the most pigmented plants to share the same genotype for that marker. If Spb is not linked to a marker, then the genotypes will be independent of phenotypes. Genotype-to-phenotype correlations were made using a computerized mapping program. This analysis defined regions of the genome containing genes that are important for determining the Spb phenotype.