Characterization of the novel maize *carbohydrate partitioning defective* mutant *P135-21B*

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Sugar synthesized by photosynthesis needs to be efficiently exported from the leaves as sucrose to feed developing tissues. There are a class of maize mutants called *carbohydrate partitioning defective* (*cpd*) mutants which overaccumulate starch and soluble sugars in their leaves. High sugar levels in the leaves result in repression of photosynthetic gene expression, chlorosis, and anthocyanin accumulation in leaves. *P135-21B*, a novel maize mutant conditioned by a semi-dominant mutation, exhibits a progressive basipetal chlorosis and starch accumulation. Three main questions were addressed: whether both starch and soluble sugars hyperaccumulate, why there is carbohydrate hyperaccumulation in mutant leaves, and what is the causal gene. In order to locate and quantify the carbohydrate accumulation in source leaves, an Iodine/Potassium Iodide stain and a quantitative measurement of sugar and starch levels using High Pressure Anion Exchange Chromatography (HPAEC) were performed. Aniline Blue staining of adult leaves suggested that the mutant phenotype may be caused by a partial blockage of leaf veins by callose accumulation. To find the rough mapping interval, pools of mutants and wild type siblings were collected and DNA was extracted for a bulked segregant analysis. A region on chromosome 1S and an interval on chromosome 7S were found to be enriched in the mutants and deficient in the wild type. In order to find the causal genes, recombination breakpoints are being screened with polymorphic markers to narrow this interval down. Neither locus is shared with any previously characterized *cpd* mutant; thus, *P135-21B* is a novel gene.