and from several \textit{sh2} and \textit{bt2} mutants indicates that each enzyme has an allele-specific Km value for glucose-1-phosphate and the level of pyrophosphorylase activity is dependent on the number of functional alleles at the \textit{Sh2} and \textit{Bt2} loci [Hannah and Nelson, 1975; 1976a]. These results suggest that \textit{Sh2} and \textit{Bt2} loci are the structural genes of the endosperm pyrophosphorylase. However, in the studies of endosperm ADPglucose pyrophosphorylase isoenzymes distinguished by heat stability [Hannah et al., 1980], two differentially heat-stable forms were reported. In wild type, 95\% of the activity exists in a form which is labile at 57°C. This form is also destroyed by electrophoretic conditions. In 16 spontaneous \textit{bt2} and \textit{sh2} mutants examined the heat labile form was reduced to a much greater extent than was the heat-stable form. These observations suggested that \textit{Sh2} and/or \textit{Bt2} maybe possibly regulatory gene(s) of enzyme. \textit{Sh2} or \textit{Bt2} may control the post-transcriptional or post-translational steps in a sequential pathway to synthesize ADPglucose pyrophosphorylase. In this hypothesis, the heat-stable form would be an intermediate to form heat-labile form, two forms would have different Km values for the glucose-1-phosphate, each step would be controlled by \textit{Sh2} or \textit{Bt2} and the wild-type revertants at \textit{Sh2} or \textit{Bt2} should have the wild-type enzyme activity. In the later experiments, \textit{Sh2} revertants induced by excision of transposable element Ds contained wild-type enzyme activity, but the Km for the glucose-1-phosphate and the allosteric