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Small-kernel ears result when *Sorbitol dehydrogenase1 (Sdh1)* is dysfunctional

(submitted by Nadia Mourad Silva <nmourad@ufl.edu>)

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Maize kernels rapidly synthesize and metabolize sorbitol via sorbitol dehydrogenase (SDH, EC 1.1.1.14). Maize SDH catalyzes the reversible interconversion of fructose + NADH \leftrightarrow sorbitol + NAD⁺. The reaction is suggested to favor sorbitol and NAD⁺ synthesis in the high-sugar, low-oxygen interior of the endosperm and possibly the reverse in other locales. However, the physiological roles of sorbitol synthesis and the fate of sorbitol in the maize kernel remain elusive. A single copy *Sdh1* gene encodes maize SDH expressed almost exclusively in the endosperm during grain fill. Preliminary analysis of a new, *Ac/Ds*-induced *sdh1* mutation indicates that it confers a small-kernel phenotype similar to that observed for a *Mu*-induced *sdh1* allele, suggesting a possible role in kernel development. We have also observed enhanced seed set in the *sdh1-Ds* mutant during multiple growing seasons but are yet to define possible mechanisms for this aspect of the *sdh1* phenotype. For physiological effects on kernel size, we hypothesize that maize SDH can aid regeneration of NAD⁺ in the low-O₂ endosperm, thus aiding glycolytic flux and helping sustain kernel growth. Characterization and biochemical analysis of *sdh1* mutants are ongoing, with preliminary results showing a ~10x decrease in sorbitol and an increase in fructose and sucrose in the mutant. The physiological and genetic effects of *Sdh1* on kernel development are being further evaluated in over-expression transgenic lines and in sweet corn, where soluble sugar levels are high. Outcomes will determine the suitability of *Sdh1* and its regulators as possible targets for genetic manipulation or metabolic engineering to alter quality and/or quantity of maize kernels.

Gene / Gene Models described: *sdh1*; Zm00001d031727

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