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GENETIC MANIPULATION OF STARCH COMPOSITION TO IMPROVE NUTRITIONAL AND TECHNOLOGICAL PROPERTIES OF DURUM AND BREAD WHEAT

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Reserve starch, the major component of wheat kernel, is constituted by two glucan polymers, amylose and amylopectin. The appropriate manipulation of amylose/amylopectin ratio results in the production of flours or semolina with novel functional characteristics. High amylose starch is particularly interesting because of its correlation with the amount of resistant starch in food, that plays a role similar to dietary fibre in the intestine with beneficial physiological effects for human health. On other hand many uses have been suggested for low amylose wheat, including as a source of blending flour to improve shelf-life and processing quality of baked and frozen products.

The absence of the starch synthases SSII or starch branching enzymes SBEIIa activities is associated to genotypes with an higher amylose content, whereas the knockout of the granule bound starch synthases I genes (GBSSI or waxy) produces a drastic decrease of amylose content. Three different strategies have been used to target SSII, SBEIIa and GBSSI proteins: 1) identification of natural mutants; 2) gene silencing by RNA interference; 3) TILLING (Targeting Induced Local Lesion in Genomes). Natural and EMS-induced mutants for SSII and GBSSI have been identified by SDS-PAGE analysis in bread and durum wheat. RNAi silencing of *SBEIIa* genes in durum wheat causes obvious alterations in granule morphology and starch composition, leading to high amylose wheat (>75%). Spaghetti produced with high amylose transgenic semolina showed improved quality characteristics, such as an increased firmness and decreased stickiness and water absorption. As these materials are very interesting for food industry, a non transgenic approach was chosen to silence *SBEIIa* genes in bread wheat. In particular TILLING analysis permitted to identify several allelic variants in *SBEIIa* genes, including putative *null* alleles containing non-sense or splice site mutations, responsible for the loss of functionality of the three *SBEIIa* homoeoalleles. Crossing activity is currently underway, in order to incorporate the three single null *SBEIIa* homoeoalleles in the same genotype.