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Poster Abstract – D.58

IS EMPTY PERICARP4 INVOLVED IN NON-SEED TISSUES DEVELOPMENT?

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Zea mais, mitochondria, embryo rescue, pentatricopeptide repeats

Empty pericarp mutants represent a class of *dek* mutants with the most severe reduction in endosperm development. They are easily recognizable in segregating mature ears because they are devoid of endosperm material and flattened by compression from the surrounding normal seeds. To elucidate the molecular basis of these mutants a gene tagging approach was undertaken in our laboratory that lead to the isolation of the *empty pericarp4* gene.

The product of this gene belongs to the pentatricopeptides repeats (PPRs) family of proteins, one of the largest gene family in plant, so called because of the presence of a degenerated 35 aminoacid repeat, the PPR motif. About 80% of the PPR proteins are targeted in organelles (60% in mitochondria and 20% in chloroplasts), and it is generally assumed that PPRs play a role in controlling the organellar gene expression. Despite their great number in the plant genome many of the PPRs so far analyzed have a non-redundant function. The *empty pericarp4* (*emp4*) gene encodes a 614 amino acid protein containing nine PPR motifs, a signal peptide for mitochondrial import and two domains with unknown function, located at the C and at N-termini respectively. Functional analysis revealed that lesions in this PPR gene are associated with specific seed developmental defects, first recognizable in the basal endosperm transfer layer (BETL), a region located in the basal portion of the seed, characterized by the early presence of transfer cells that facilitate nutrients import into the maize kernel. Moreover the presence of a functional *emp4* in the maize kernel has been correlated with the accumulation of three mitochondrial transcripts, *rps2A, rps3* and *mttB.*

emp4 transcription occurs, even though at a very low level, in most plant tissues. Because of this observation we tried to estabilish if the *emp4* gene product, besides exerting a specific role during seed formation, is involved in other developmental events.

Homozygous *emp4* mutant embryos are retarded in their growth and unable to germinate. To analyze EMP4 function during postembryonic stages, immature embryos have been excised from the kernel and cultured on synthetic media. Following the embryo rescue approach we obtained homozygous mutant seedlings, whose genotype has been ascertained through a PCR based strategy. These seedlings exhibit delayed growth and are unable to reach reproductive maturity. Data will be presented on the morphological and molecular analysis of mutant roots, leaves and stem tissues. Changes in the mitochondrial and chloroplast population have been highlighted from the comparison of wild-type and mutant tissues by means of transmission electron microscopy. In addition, the expression of a sub set of mitochondrial genes has been investigated in the same

tissues by semi quantitative RT-PCR. The effect of the mutation on the expression of these genes in non seed tissues will be discussed.