

ANALYSIS OF MITOCHONDRIAL GENES AND THE ASSOCIATION WITH CYTOPLASMIC MALE STERILITY- RNA EDITING**Hideaki Suzuki****Jinfa Zhang****New Mexico State University****Las Cruces, NM****James McD. Stewart****University of Arkansas****Fayetteville, AR****Abstract**

Cytoplasmic male sterility (CMS) is a maternally inherited trait in more than 150 higher plants, resulting in failure to produce functional pollen. The CMS system is widely used in hybrid breeding to produce F1 hybrid crops on a commercial scale to utilize heterosis, i.e., hybrid vigor. CMS is caused by the dysfunction of mitochondrial DNA genes encoding for ATP subunits (atp) or cytochrome oxidases (cox). However, the molecular mechanism of CMS in cotton is currently unknown. RNA editing, one of the post-transcriptional processes leading to C-to-U change in nucleotide sequence of RNA, is implicated in some CMS systems, and is essential to restore functionality of protein. The objective for this study was to detect RNA editing sites in mitochondrial genes, atp1, 4, 6, 8 and 9, and cox1, 2 and 3 of maintainer (with AD1 cytoplasm and non-functional recessive restorer rf2 gene), CMS (with CMS-D8 cytoplasm and non-functional recessive restorer rf2 gene) and restorer (with CMS-D8 cytoplasm and functional dominant restorer Rf2 gene) lines, and to identify editing efficiency differences between CMS-D8 and its restorer line. PCR and RT-PCR were conducted using mitochondrial gene specific primers from the three lines and followed by cloning and sequencing. The sequences were analyzed by CluatalW and CAP3. Fifty-five C-to-U and two U-to-C RNA editing sites were identified in the eight sequenced mitochondrial genes. The majority of amino acid changes due to RNA editing causes alternation of hydrophilicity to hydrophobicity. Seven editing efficiency differences between CMS-D8 and its restorer line were found which were due to the restorer gene Rf2. A few nucleotide replacements were also identified between CMS-D8 and AD1 cytoplasms. However, association between RNA editing and CMS in mitochondrial genes studied, i.e., atp1, 4, 6, 8 and 9, and cox1, 2 and 3 is not clear at present. Further studies will be needed.