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THE CITRATE LYASE REGULATES CELL DIVISION DURING *DROSOPHILA* MALE MEIOSIS

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The Citrate Lyase (ACL) is the main cytosolic enzyme that converts the citrate exported from mitochondria, in acetylCoA and ossalacetate. As Acetyl-CoA is an essential substrate for the biosynthesis of cholesterol and long-chain fatty acids, ACL acts as critical enzyme for the de novo synthesis of a wide range of complex cellular lipid. In addition, ACL provides acetyl-CoA for histone acetyltransferase in the cell nuclei. We have isolated a P-element induced male sterile mutation in the Drosophila *DmACL* gene that encodes the ortholog of human ACL, suggesting a role of ACL during male meiosis and spermatogenesis in fruit flies. *in vivo* analysis of mutant testes revealed the presence of multinucleate spermatids and micronuclei. More than 80% (n= 120) of mutant spermatids exhibited an irregular association between the nebenkern, a mitochondrial derivative, and nuclei indicating that loss of ACL gives rise to a failure of cytokinesis in male meiosis. In addition, 20% of spermatids exhibited also nuclei of different sizes, suggesting that chromosome segregation is also affected upon depletion of ACL. Immunolocalization of a-tubulin and the centriole component Spd2 revealed the presence of multipolar spindles during both meiotic divisions. Particularly, we found an high proportion of anaphase I cells (80%; N=50) with either bipolar, tripolar ("bugs bunny-like" configurations) or quadripolar spindles that contained four distinct nuclei each denoting a cytokinesis defect occurring during primary spermatocyte formation. DAPI staining of mutant meiotic chromosomes showed also the presence of either lagging chromosomes and chromatin bridges which are likely to give rise to micronuclei in spermatids. Here we present data suggesting that depletion of ACL causes 1) cytokinesis failure because of alterate membrane synthesis in male meiotic cell division and 2) chromosome segregation defects as consequence of an umbalanced acetylation status of chromatin.