

MALE STERILE AUTOSOMAL MUTATIONS AFFECTING DYNEIN EXPRESSION IN DROSOPHILA MELANOGASTER

R. Piergentili, S. Bonaccorsi, *C. Mencarelli and M. Gatti

Dept. of Genetics and Molecular Biology, Univ. La Sapienza, Roma, and

*Dept. of Evolutionary Biology, Univ. of Siena, Siena, ITALY

In *Drosophila melanogaster*, deletions of the kl-3 fertility factor of the Y chromosome result in the simultaneous loss of the outer dynein arms from the sperm flagellar axoneme and of a single polypeptide thought to be a dynein heavy chain. In addition, deletions of the kl-3 region cause the absence of a prominent lampbrush-like loop from primary spermatocyte nuclei. These results have suggested that the kl-3 fertility region contains an axonemal dynein gene and that the intranuclear lampbrush-like structure represents the cytological manifestation of its transcription. However, since no axonemal dynein sequences have been as yet identified within this region, it is also conceivable that this Y-linked locus only regulates dynein rather than containing a dynein structural gene.

In order to elucidate the functional role of the kl-3 locus in the assembly of *Drosophila* flagellar axonemes we isolated and characterized 4 male sterile autosomal mutations that specifically affect the formation of the kl-3 loop in primary spermatocytes. All these mutations have been genetically mapped; two of them, ms(2)HA30 and ms(2)HB108, severely reduce the size of the kl-3 loop, while the other two, ms(3)HB223 and ms(3)HB267, completely suppress the formation of this structure.

Males carrying any of these 4 mutations were characterized for the presence of dynein polypeptides by PAGE-SDS of testes extracts, and for the presence and normality of the outer dynein arm by EM analysis of sperm tail sections. These studies showed that mutations that suppress the kl-3 loop strongly affect both the expression of at least three dynein polypeptides and the correct assembly of the axoneme.