Mutations affecting the kl-3 loop of the Drosophila melanogaster Y chromosome.

Piergentili R.¹, Mencarelli C.², Raffa G.¹, Gatti M¹. and Bonaccorsi S¹.

¹Dipartimento di Genetica e Biologia Molecolare, Università di Roma "La Sapienza", P.le A. Moro 5, 00185 Roma, Italy

²Dipartimento di Biologia Evolutiva, Università di Siena, Via Mattioli 4, 53100 Siena, Italy

Deletions of the kl-3 fertility factor of the *Drosophila melanogaster* 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 (1). In addition, deletions of the kl-3 region cause the absence of a prominent lampbrush-like loop from primary spermatocyte nuclei (2). These and other recent results (3) 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, based on its peculiar molecular and cytological features, it was also suggested that the kl-3 loop can fulfill an unconventional role, probably by binding proteins necessary for the proper assembly of the sperm tail (4).

In order to elucidate the functional role of the kl-3 locus we isolated and characterized 4 autosomal male sterile mutations that suppress the formation of the kl-3 loop. Complementation tests revealed that these mutations identify two loci that were named <u>Suppressor of kl3loop-1</u> (Suk3l-1) and <u>Suppressor of kl3loop-2</u> (Suk3l-2), respectively. mutant males were then examined both for the presence of dynein polypeptides by PAGE-SDS of testis extracts, and for the trasence and normality of the axonemal dynein arms by EM analysis of sperm tail sections. Both Suk3l-1 and Suk3l-2 do not impair the synthesis of the kl-3 putative dynein subunit described by Goldstein et al. (1). Surprisingly, however, the stability of four high molecular weight polypeptides is simultaneously affected in each mutant, together with the regular assembly of the axonemal dynein arms, that are either absent or strongly reduced.

References

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