Glätzer, K.H. and P.-M. Kloetzel.* University of Düsseldorf and *University of Heidelberg, FR Germany. Antigens of cytoplasmic RNP particles of **D.melanogaster** can be localized on distinct Y chromosomal structures in spermatocytes of **D.hydei.**

RNP particles can be isolated from the cytoplasm of Drosophila cells. The particles have been characterized in detail both biochemically and structurally (Schuldt & Kloetzel 1985): the particles consist of 16 polypeptides within the MW range 23000-35000 dalton and contain three small RNAs with as yet unknown function. In the electron microscope the RNP particles have a ring like appearance with an outer diameter of 12 nm and an inner core of 3 nm. With the help of antibodies raised against the different particle proteins it was shown that at least one of the small heat shock proteins of Drosophila must be a genuine part of the 19s particles.

Despite the cytoplasmic location of the RNP particles their constituent antigens are found specifically concentrated on Y chromosomal structures in male germ cells, i.e., in nuclei of primary spermatocytes of Drosophila. In particular, antibody Dm 28K2 reacts very strongly with the Y chromosomal "pseudonucleolus" and "clubs" in spermatocyte nuclei of **D.hydei** (Fig. 1a-c). This specificity can now be used to look at different translocation stocks in order to test for example whether or not the translocated Y fragment still carries the segment responsible for the formation of the "clubs". As known from light microscopical observations, the morphology of a single Y chromosomal structure changes considerably if the whole complement of the Y chromosome is not present in the spermatocyte nucleus. This can also be seen in the electron microscope. The ultrastructure of RNP in spermatocyte nuclei in certain translocation stocks indicates that the presence of a Y section per se is not sufficient to guarantee the normal spatial relation of RNP structures to the Y chromatin (Glaetzer et al., in prep.). This is also evident by indirect immunofluorescence with the antibody Dm 28K2. Despite the presence of the "clubs" the morphology and distribution of this nuclear compartment is greatly altered in these strains (Fig. 1d,e). Even in translocation stocks which by cytogenetical data should carry the same Y chromosomal segment, the "clubs" differ characteristically (data not shown). Another example is stock 701/17 supposed to contain mutated "nooses", i.e., "granular nooses" (Hess 1970). As seen in Fig. 1f the spermatocyte nucleus clearly shows "club"-specific material. Thus, this stock should be reinvestigated with respect to the translocated Y chromosomal segment.

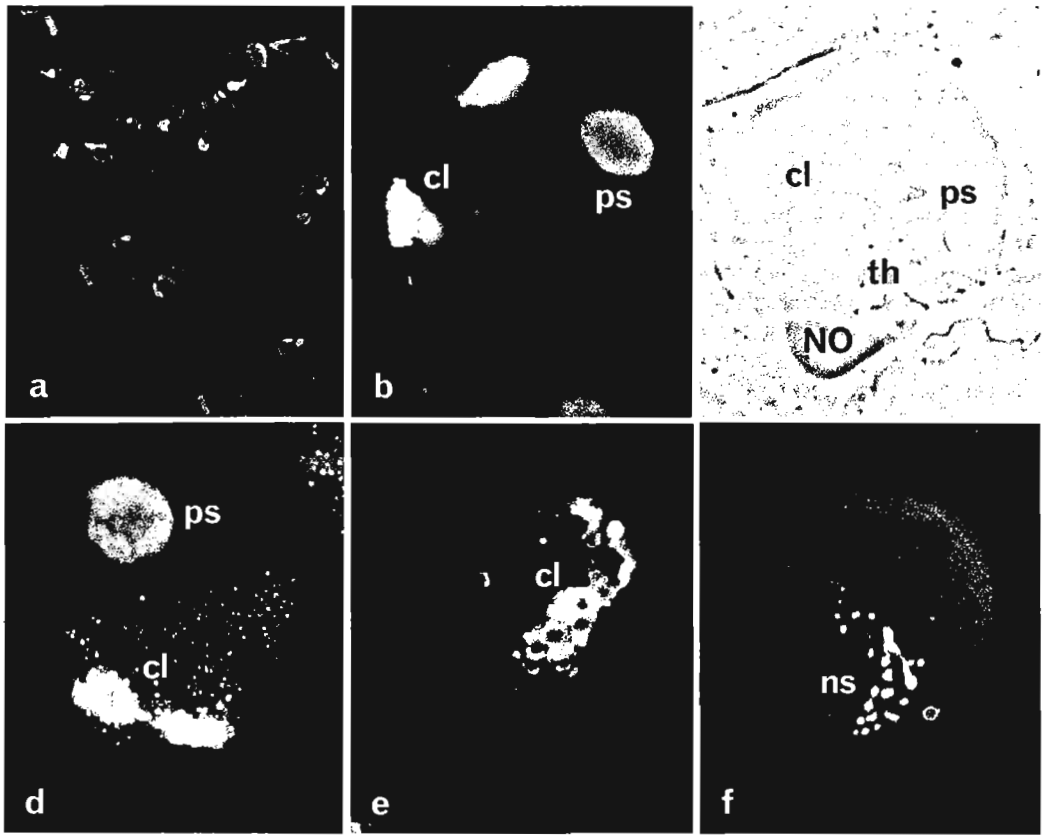

Figure 1. Localization of antigen Dm 28K2 in nuclei of primary spermatocytes by indirect immunofluorescence. (a) squash of wild type spermatocytes, survey photograph to show the identical immunoreaction in several nuclei; (b) a wild type nucleus enlarged to show the specific reaction of antibody Dm 28K2 with distinct Y chromosomal structures; (c) the same nucleus as depicted on b in phase contrast; (d) nucleus of a genotype lacking "tubular ribbons"; (e) nucleus with a Y fragment carrying only "nooses" and "clubs"; (f) nucleus with a Y fragment supposed to carry "granular nooses" **cl**: clubs, **ns**: nooses, **ps**: pseudonucleolus, **th**: threads, **NO**: nucleolus organizer. Magnifications: a) x250, b-f) x1125.

The application of specific antibodies against gene products that play a role in developmental processes, here spermiogenesis in Drosophila, offers a good opportunity for elucidating the role of particular components involved in that process (cf. Glaetzer 1984; Melzer & Glaetzer, this issue). For example, there are indications that the cytoplasmic RNP particles have a regulatory function (Kloetzel et al., in prep.). This is paralleled by the localization of similar polypeptides on specific Y chromosomal formations known to exert regulatory functions in spermiogenesis.

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References: Glaetzer, K.H. 1984, Mol. Gen. Genet. 196:236-243; Hess, O. 1970, Mol. Gen. Genet. 106:328-346; Schuldt, C. & P.-M. Kloetzel 1985, Devel. Biol., in press.

González, A. and J.L. Ménsua. University of Valencia, Spain. Allelic rates and population sizes of two populations of **D.melanogaster** from cellar and vineyard.

Captures of **D.melanogaster** were carried out simultaneously in two sites, one cellar and one vineyard, both located in Requena (Valencia) in the east of Spain. In a previous study of these two populations, which are approximately four kilometres away, the relative viabilities of heterozygotes and homozygotes, frequency of lethals and D:L relation of third chromosomes were compared (Gonzalez & Mensua 1983). In this study the allelism rates of lethal third chromosomes from vineyard and cellar are presented, both intra- and interpopulations.

The effective size (N_e) of both populations was estimated according to Nei (1968). This formula assumes that the degrees of dominance of lethal genes and the mutation rates to lethals (u) per locus are the same for all loci:

$$\hat{N}_e = (1-\hat{I}_g) / 4(\hat{I}_g U-u) \ ,$$

where: I_g stands for the allelism rate of lethal genes. Can be estimated by:

$$I_g = \ln(1-I_C Q^2) / (\ln (1-Q)^2) \ .$$

I_C stands for the allelism rate of lethal chromosomes. Q is the frequency of the lethal chromosomes. U is the total lethal mutation rate; Wallace (1968) estimated this for third chromosome 0.005. u is the lethal mutation rate per locus; values of 10^{-5} and 0.20×10^{-5} were used in our calculation; the former estimate comes from the number of lethal producing loci per second chromosome (n=500); this value is assumed also for the third chromosome. The latter estimate is based on n=2.400 (Judd et al. 1972).

The results of allelism test are shown in Table 1. Low frequencies of allelism are observed in the two populations and between populations. There are no significant differences among the three estimates (5% level). Clusters of allelic lethals did not exist in any of the two populations.

The part of the allelism observed which is caused by chance mutations and the part due to consanguinity were estimated in the vineyard and cellar populations according to Wallace (1966), Table 2.

The allelism due to chance mutations was similar in both populations. The greater frequency of allelism observed in the cellar compared to the vineyard, although the difference between these frequencies is not significant, can be attributed essentially to the greater consanguinity inside the cellar habitat.

Table 1. Allelism tests of lethal third chromosomes from cellar and vineyard populations.

Populations crossed	No. of lethal chromosomes	No. of crosses completed	No. of allelic crosses	Frequency of allelic crosses
cellar x cellar	38	703	6	0.00853±0.00347
vineyard x vineyard	40	780	5	0.00641±0.00286
cellar x vineyard	79	1520	5	0.00329±0.00147

Table 2. Estimated values of allelic frequencies of lethals due to chance mutations and consanguinity.

	I_C	I_N	I_F
Vineyard			
n = 500	0.0064	0.0027	0.0037
n = 2400		0.00057	0.0058
Cellar			
n = 500	0.0085	0.0026	0.0059
n = 2400		0.00055	0.0079

I_C = Allelism frequency of lethal chromosomes; I_N = a.f. due to chance mutations; I_F = due to consanguinity.