plained. Also, cytoplasmic interference cannot be excluded since differences in albumin, globulin, gliadin and glutenin contents have been found to be the consequence of the source of cytoplasma in reciprocal triticale populations[11].

Among the 5 isoenzymes in triticale, besides $\varrho\varrho$ homodimers with the slowest mobility, there are 3 heterodimer types containing ϱ subunits: $\alpha\varrho$, $\beta\varrho$ and $\delta\varrho$. This ability to aggregate would first of all presuppose similar structures due to related genes on at least partially homoeologous chromosomes (beta-arm of chromosome 4 in wheat and chromosome 4R/7R in rye[3]). Minor evolutionary differences in the ADHs of wheat and rye should only be expressed immunologically. The results are consistent with the expectation. In figure 2 summarized recordings of all triticale bands (T), wheat bands (W) and the single rye band (R), incubated with antiserum against wheat-ADHs, are given in percent of the activities found in triticale, wheat and rye extracts after having been incubated with control serum (C). ADH activity of rye is scarcely affected by anti-wheat-ADH. Therefore, it seems to be proved that ADH subunits of wheat and rye can aggregate to form enzymatically active heterodimers in triticale in spite of the evolutionary divergence of their genes.

11 S. L. K. Hsam and E. N. Larter, Can. J. Genet. Cytol. *16*, 529 (1974).

Interorder transfer of mycoplasmalike microorganisms between *Drosophila paulistorum* and *Ephestia kühniella*. II. Numbers of MLO and sterility

F. J. Gottlieb, R. Goitein[1], L. Ehrman[2] and S. McAvoy[2]

Department of Life Sciences, University of Pittsburgh, Pittsburgh (Pennsylvania 15260, USA), and Division of Natural Sciences, State University of New York, Purchase (New York 10577, USA), 21 March 1977

Summary. A successful attempt was made to culture the mycoplasmalike microorganism causing semispecific hybrid male sterility in *Drosophila paulistorum* utilizing *Ephestia kühniella* as the intermediate host. Data gleaned from this passage indicates that the induction of sterility depends upon the quality not the quantity of infectious intracellular symbionts.

Genetic relationships between a host and its well established endosymbiont are difficult to study under normal conditions. Occasionally controls break down and the symbiont becomes a pathogen. When this happens we are able to gain insight into the modes of action of the symbiont in its 2 expressions. The involvement of mycoplasmalike organisms (MLO) in the male sterility of *Drosophila paulistorum* intersemispecific hybrids is known[3]. Each of the 6 semispecies possesses an MLO strain, possibly unique, which normally has no deleterious effect on its own host. The symbiont is present in the gonads of both sexes and is passed from generation to generation through the egg cytoplasm[4]. Male sterility results from the introduction of a strain of MLO into a host with a foreign genetic background, either by hybridization or by injection of testis-extracts[5,6]. Genetic incompatibility was thought to lead to unrestrained growth of the symbiont. Thus the sterility was assumed to be the direct result of rapid proliferation of MLO within sperm cysts. For this reason, and because MLO transfer is through the egg, it was concluded that the gonads were not only the target tissue in hybrid males but also the primary tissue of localization in both sexes[3]. Drosophila paulistorum MLO can be transferred to and proliferate in Ephestia kühniella Zeller (the Meditarranian meal moth)[7]. In this new host the MLO are pathogenic and can be further passaged in Ephestia or passed back to Drosophila without loss of host-strain recognition specificity[8]. The pathogenicity in *Ephestia*, as measured by the time and mode of dying of extract recipients, is dose dependent. It was expected that testis-extracts of pure strain Drosophlia would contain fewer MLO than those of hybrids because of the normal control of symbiont proliferation in its own host strain. This was not the case. Testis-extracts of Mesitas (M, Andean semispecific) strain or of Santa Marta (SM, Transitional semispecific) strain were as potent as those of the 2 reciprocal male hybrids: (SM♀ × M♂) and (M♀ × SM♂).

Heads of hybrids also proved to be highly pathogenic with an extract of 1 head/ml being equivalent to approximately 2–4 testes/ml. The assumption that MLO are mainly localized in the gonads is no longer tenable. The presence of MLO in heads provides an easy way to estimate the relative distribution and concentration of MLO in the pure strains of *Drosophila* as compared with one another and with hybrids. Extracts of heads of a representative pure strain from each of the 6 semispecies were injected into Ephestia larvae. All extracts were at least as higly pathogenic as the hybrid extracts. There appear to be as many MLO present in pure strain heads as in hybrid heads. Indeed, 2 of the extracts (Brazil, Amazonian semispecies, and Llanos, Interior semispecies) were more virulent than the hybrid extracts. We interpret this to indicate that larger numbers of MLO are present in these 2 pure strains, although we cannot as yet rule out the possibility that Brazil and Llanos MLO are more virulent than those of the other 4 semispecies (including Orinocan and Centroamerican ones).

Neither of these latter observations would be expected under the earlier assumptions. These results therefore raise serious questions about the relationship between the number of MLO and hybrid male sterility in *Drosophila paulistorum*. It can no longer be assumed that sterility in hybrids is due solely to the presence of larger numbers of MLO than is normal. Sterility is not just the product of mechanical problems caused by the massive numbers of MLO. Rather, semispecific male hybrid sterility must also involve both host-symbiont recognition factors and a tissue specific response in the testes to the presence of the 'wrong' MLO.

1 On leave from the Department of Genetics, The Hebrew University, Jerusalem (Israel).
2 Division of Natural Sciences, State University of New York, Purchase (NY 10577, USA). Reprint requests to L.E.
3 L. Ehrman and R. P. Kernaghan, in: Pathogenic Mycoplasmas, p. 227. Associated Scientific Publishers, Amsterdam 1972.
4 L. Ehrman and S. Daniels, J. Invert. Path. *24*, 14 (1974).
5 L. Ehrman, H. Levene and L. Ramos, Aust. J. biol. Sci., in press (1977).
6 D. L. Williamson and L. Ehrman, Genetics *55*, 131 (1967).
7 F. J. Gottlieb and L. Ehrman, Genetics *83*, 27 (1976).
8 F. J. Gottlieb, R. Goitein, L. Ehrman and B. Inocencio, J. Invert. Path. *30*, 140 (1977).