of flies died during the experiment, less than 40% on 0.0025% DDT and less than 10% on 0.001% DDT. The results are shown in Fig. 2. Also in this experiment a significant increase in the activity of G6PD and 6PGD is found in flies on DDT, though at the lowest concentration only the males respond. In spite of some differences in reaction between males and females it is evident that DDT treatment can strongly increase the activity of G6PD and 6PGD in adults. Preliminary experiments suggest that this also holds for larvae.

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Three mutations blocking early steps in Drosophila oogenesis: fs(4)34, fs(2)A16, and fs(1)231M.

The fs(4)34 mutation was recovered by B. Hochman from a male collected in December of 1963 from a natural population of Drosophila melanogaster living in Lake County, Florida. Hochman (1972) reported that the mutant resided on the 4th chromosome, and it is still the only female sterile gene known for this microchromosome. Two major deficiencies, Df(4)M and Df(4)G, allow the right arm to be partitioned into three unequal parts (Hochman, 1974). Hochman showed that fs34 is not included in either deficiency, and therefore it probably resides somewhere between the middle of subdivision 102B and the beginning of subdivision 102E (see King, 1975, his Fig. 1). The heterozygotes used for the maintenance of the stock population are of genotype fs(4)34/ci^{D}. The fs(2)A16 mutation was induced by Bakken (1973) using EMS. She studied ovarian whole mounts from homozygotes and concluded that the ovarioles lacked clear cut germaria. The mutation has not yet localized on chromosome 2. Heterozygotes have the genotype fs(2)A16/SM1.

The results of our electron microscopic studies are summarized in the accompanying illustration of sections from 2 day ovaries (Fig. 1). The fs34 ovariole begins with a normal terminal filament which is anchored to the tubular epithelial sheath. The anterior end of the germarium contains abnormally large numbers of oogonial cells and lacks clusters of cysto-

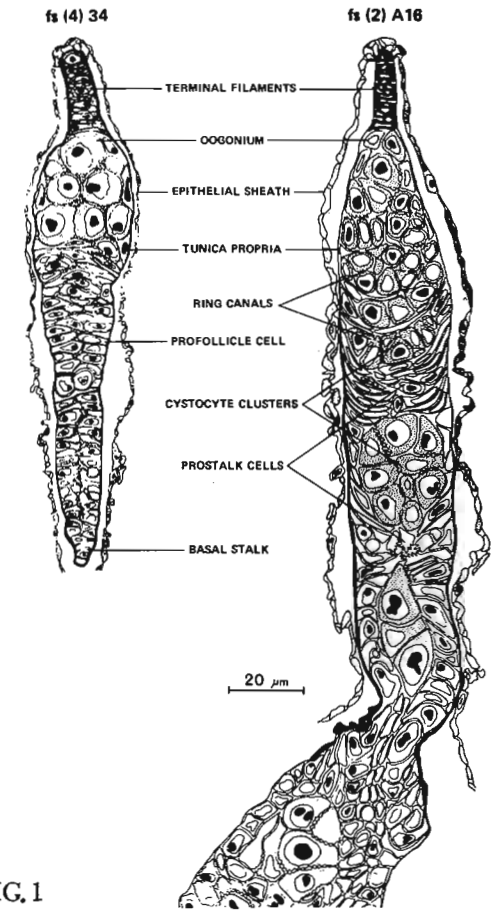

FIG. 1

FIG. 2

cytes. The remainder of the germarium consists of a solid cylinder made up of profollicle cells which ends blindly as a basal stalk. The entire structure is coated by an acellular tunica propria. In the fsA16 germarium clusters of cystocytes are abundant, and the ring canals that connect them are often included in sections. Clustered cystocytes are surrounded by wedge-shaped follicle cells. However, adjacent clusters are not connected by inter-follicle stalks, and consequently a conventional moniliform vitellarium is not observed. The fault seems to lie with the prostalk cells which fail to interdigitate. The posterior end of the ovariole is filled with fusing follicles. Thus in the case of the fs34, oogenesis appears to be blocked at the point where oogonia are converted to cystoblasts. The developmental block is later in fsA16 ovaries and seems to involve the mesodermal cells at the base of each germarium. Since these cells fail to form the stalks that allow egg chambers to bud off continuously from the germarium, the mutant has been nicknamed "stalkless".

The fs(1)231 mutation was induced by Gans et al. (1975). The fs(1)14-97 mutation was recovered subsequently by J.D. Mohler using previously described techniques (Mohler, 1977). King et al. (1978) reported that fs231 belongs to the ovarian tumor class. Cystocytes require the product of the $fs231^{+}$ gene for cytokinesis to be arrested. Cystocytes that cleave completely undergo supernumerary divisions and generate ovarian tumors. The B138L/B170R deficiency includes fs231. According to Kambysellis (1977) females homozygous for fs14-97 have rudimentary ovaries and accumulate large amounts of vitellogenin in their hemolymph.

The cross fs231 v^{24}/FM3 ♀ X y cv fs14-97 v f ♂ produces sterile daughters of genotype y cv fs14-97 v f / + + fs231 v^{24}+. The cross B138L/B170R/FM7 ♀ X y cv fs14-97 v f ♂ produces fs14-97/B138L/B170R females that are also sterile. Fig. 2 shows a light micrograph of a section through the ovary of an 11 day old 231/14-97 female. Tumors containing hundreds to thousands of cystocytes are present. Thus fs231 and fs14-97 are alleles, and we refer to them as fs231G and fs231M, respectively.

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Kiss, I. and J. Szabad. Institute of Genetics, Biological Research Center of the Hungarian Academy of Sciences, Szeged, Hungary. Characteristics of some new X-linked pupal lethals of D. melanogaster.

A detailed study on lethal mutants which show a normal larval development but a lack or a delay in puparium formation has been made recently in our laboratory (Kiss et al. 1978). Similar experiments were made also with other mutants having a significantly longer than normal larval development time. In these experiments we made a general characterization of the mutant phenotypes and tested the autonomous expression of the non-pupariating character in gynander mosaics and by implanting wild-type ring glands into mutant larvae. For the technical details, see Kiss et al. (1978). The mutant chromosomes were marked with y and w.

Table 1. General characteristics of the mutants

Mutant	Developmental time[1] (in days)	Frequency of puparium formation (%)	Imaginal discs		Ring gland size[2]	Metamorphotic capabilities			Map position
			Size	Folding		Prepupal molt	Pupal molt	Histolysis	
ℓ/1/1-43	10;12	25	very small	ø	small	+	ø	+	26.3
ℓ/1/1-45	7;8	33	very small	ø	small	+[3]	+[3]	+	58.7
ℓ/1/1-48	8;10	4	small	undeveloped	small	+[3]	+[3]	+	66.4
ℓ/1/1-74	11;15	2	small	undeveloped	normal	+	+	+	18.5

[1]Days until reaching the size of a mature wild-type larva; days until the beginning of puparium formation.
[2]At the time of puparium formation.
[3]Data refer to the abdomen only. No differentiation of the head and thorax was observed (see also Fig. 1)
Abbreviations used: + = yes; ø = no.