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.

References: Bakken, A.H. 1973, Dev. Biol. 33:100-122; Gans, M., C. Audit and M. Masson 1975, Genetics 81:683-704; Hochman, B. 1972, DIS 48:17; 1974 Cold Spring Harbor Symp. Quant. Biol. 38:581-589; Kambysellis, M.P. 1977, Am. Zoologist 17:535-549; King, R.C. 1975, Handbook of Genetics 3:625-652; King et al. 1978, Int. J. Morphol. Embryol. 7:359-375; Mohler, J.D. 1977, Genetics 85:259-272.

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.

General characteristics of the mutants are summarized in Table 1. Common features of these mutants were the delayed larval development, the rudimentary appearance of their imaginal discs and the formation of abnormal puparia. The puparia never contracted properly, and tanning and sclerotization of the cuticle was unequal and insufficient. The undeveloped imaginal discs seemed to prevent the normal differentiation of head and thorax regions in *l*/1/1-45 and *l*/1/1-48 puparia: the pupal molt occurred only on the abdomen and a small, undifferentiated rudiment was found in the place of the head and thorax (Fig. 1). *l*/1/1-74 larvae had an extremely small amount of fat body.

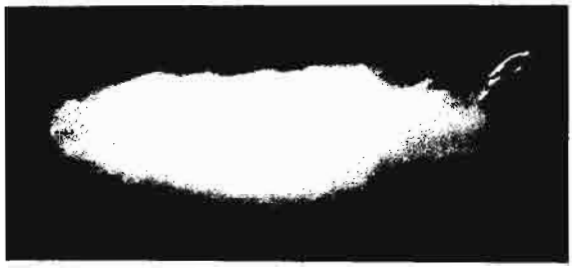

Fig. 1. Four day old "pupa" of *l*/1/1-45. The abdomen is properly formed and covered by prepupal and pupal cuticles. There are no identifiable structures differentiated in the head and thorax region and the degenerating rudiments have no cuticle.

Autonomous expression of the mutant phenotype was studied in gynander mosaics and in ring implantation experiments. The results of these tests are shown by Tables 2 and 3.

As for the action of the mutant genes, the observations suggest the following interpretation: the genes are already expressed during the larval period; therefore, the larval development is slowed down in the mutants (Table 1). The lack of larval-puparial mosaics (Table 2) suggests that the delayed pupariation character is not autonomous in the larval epiderm, probably being an indirect consequence of the mutant gene action in all the four mutants.

The implantation of normal ring glands (the main source of ecdysone in the larva) into mutant larvae caused a significant "acceleration" of puparium formation only in the case of *l*/1/1-43 (Table 3); this probably means that in this mutant the ecdysone concentration is too low to induce pupariation of the mature larva. However, it cannot be an "ecdysoneless" mutant; otherwise it should be lethal around the first larval molt (Garen et al. 1977). In the other two mutants tested the cause of the delay in pupariation is not simply a low, subthreshhold ecdysone titre.

The lack of the adult gynanders in all the mutants (Table 2) suggests that viable mosaic combinations are very rare or missing and might be explained by assuming the lethal focus being very large or multiple.

Table 2. Results of the gynander mosaicism test

Mutant	$\frac{y\ w}{R/1/2}$ gynander mosaics			
	Found		Expected[1]	Viability[2]
	Adult	Larval-Puparial		
l/1/1-43	0	0	34	<0.029
l/1/1-45	0	0	61	<0.016
l/1/1-48	0	0	46	<0.022
l/1/1-74	0	0	48	<0.021

[1] Number of the adult gynanders expected = 1.7 X no. of $\frac{Binsn}{R/1/2}$ gynanders found (Kiss et al. 1978).

[2] Gynander viability < $\frac{1}{\text{no. of gynanders expected}}$

Table 3. Effect of implanting wild type ring glands into mutant larvae

Mutant host	No. of larvae injected		Time (in days) till the beginning of puparium formation		% pupariation in operated animals until the beginning of spontaneous pupariation in the control	% pupariation within 5 days following the operation	
	Experimental	Control	Experimental	Control		Experimental	Control
l/1/1-43	17	16	1	5	59	59	6
l/1/1-45	39	26	1	1	-	15	4
l/1/1-48	44	28	2	4	14	16	11

References: Garen, A., L. Kauvar and J.-A. Lepesant 1977, Proc. Nat. Acad. Sci. (Wash.) 74:5099-5103; Kiss, I., J. Szabad and J. Major 1978, Molec. Gen. Genet. 164:77-83.