phenotypes defines a total of 61 loci on the second chromosome, 48 (79%) of which are represented by more than one allele. The arithmetic average is 4.5 alleles per locus. The distribution of allele frequencies is described in Fig. 2. It is note-worthy that the average allele frequency is significantly lower for loci with head defects as their predominant feature than for all other loci (see Discussion). Of the 14 "head" loci, 3 (21%) have more than two alleles, while the corresponding percentage for the 47 other loci is 67%. Of the latter, 83% have more than one and 17% more than seven alleles per locus. Mutants at three complementation groups are not embryonic lethal.

Genetic mapping

Members of each complementation group as well as the single mutants were mapped by recombination with visible markers as described in Materials and methods. Whenever possible, the back-cross was performed with an independent allele in order to complement other lethal functions which might have been induced simultaneously in the heavily mutagenized chromosome. In most cases the lethal function as well as the phenotype was mapped and the two sets of data were usually in good agreement. Mapping the lethal function in the 17 mutants with no known alleles showed that all but one had more than one lethal mutant per chromosome. In these cases, the mapping data are based solely on mapping of the mutant phenotype.

In order to assess allelism of some of our mutants with previously established loci, we inspected the lethal phenotypes of several dominant and recessive lethal mutants on the second chromosome as well as second-chromosomal deficiencies (Table 1). Some of these showed an embryonic phenotype and, in cases in which allelism with members of our complementation groups was likely to be based on phenotype and map position, this was tested by complementation. Our mutant collection contains alleles at the previously known loci *Dopadecarboxylase* (Wright et al. 1976a), *engrailed* (Kornberg 1981), *Krüppel* (Gloor 1950; Wieschaus et al. 1984b), *Star* (Lindsley and Grell 1968), *wingless* (Babu 1977), *decapentaplegic* (Spencer et al. 1982), *crinkled* (Lindsley and Grell 1967; *l(2)br27* of Ashburner et al. 1982), and *snail* (*l(2) br28* of Ashburner et al. 1982). For the cytological location of some of the complementation groups, complementation tests were performed between chromosomes bearing a deficiency for the second chromosome and one or two members of complementation groups mapping within 10 map units of the deficiency. We tested 42 deficiencies covering a total of about 30% of the second chromosome and the approximate cytological location of 24 of the 61 loci was determined. Further, a number of translocations (Nüsslein-Volhard and Kluding, unpublished work), in which larger parts of the second chromosome segrated with the Y or third chromosome were tested as to whether the deficiency segregant uncovered the mutant phenotype. Since individuals hemizygous for large chromosomal deficiencies are usually embryonic lethal, showing phenotypes overlapping certain phenotypic classes of zygotic point mutants, only mutants with striking and unique phenotypes could be tested. The cytology of the chromosomal aberrations used is listed in Table 1 and illustrated in Fig. 3. The map positions of the 61 loci are indicated in Fig. 3 and Table 3.

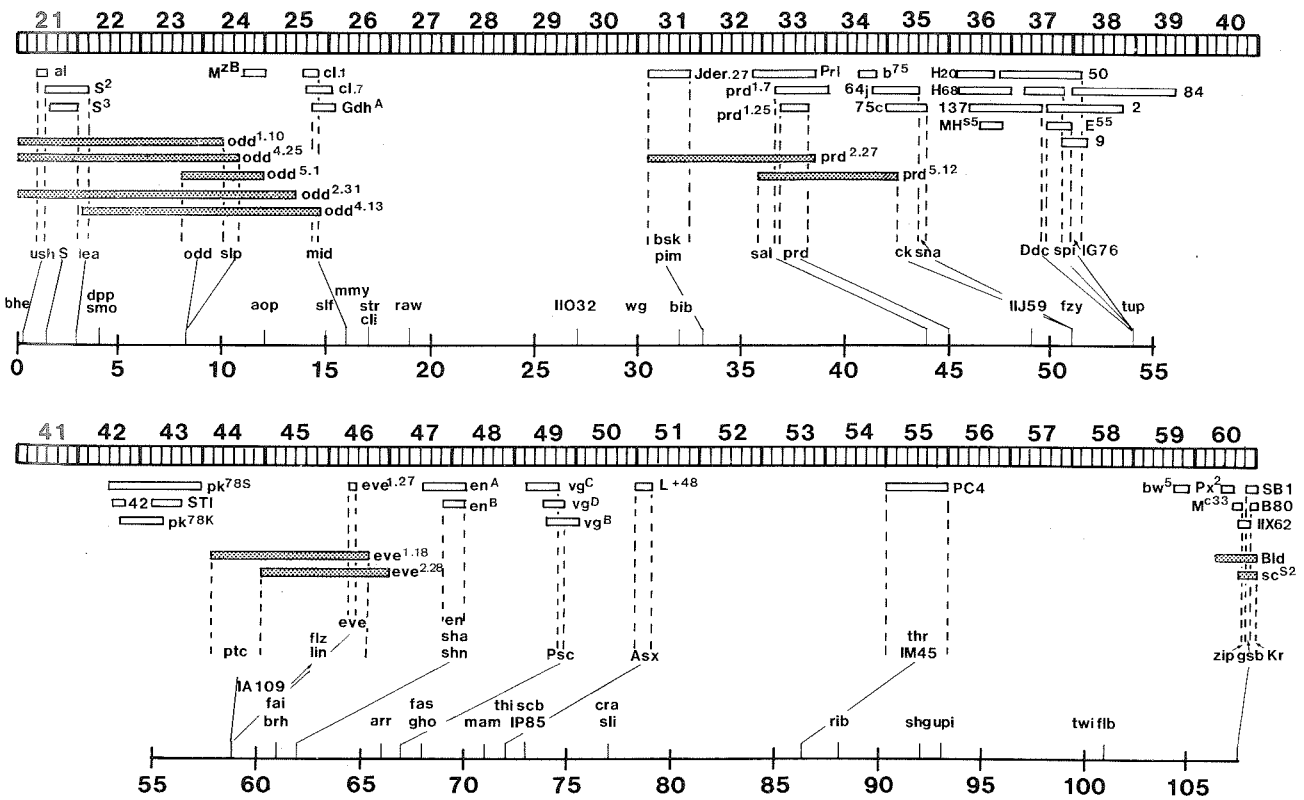


Fig. 3. A simplified map of the second chromosome indicating map position and cytological localization of the 61 loci. Deficiencies are represented by *open bars*, translocations by *shaded bars*. Cytologically undefined loci are shown directly above the genetic map. The more detailed cytological description of the chromosomal aberrations may be found in Tables 1 and 3

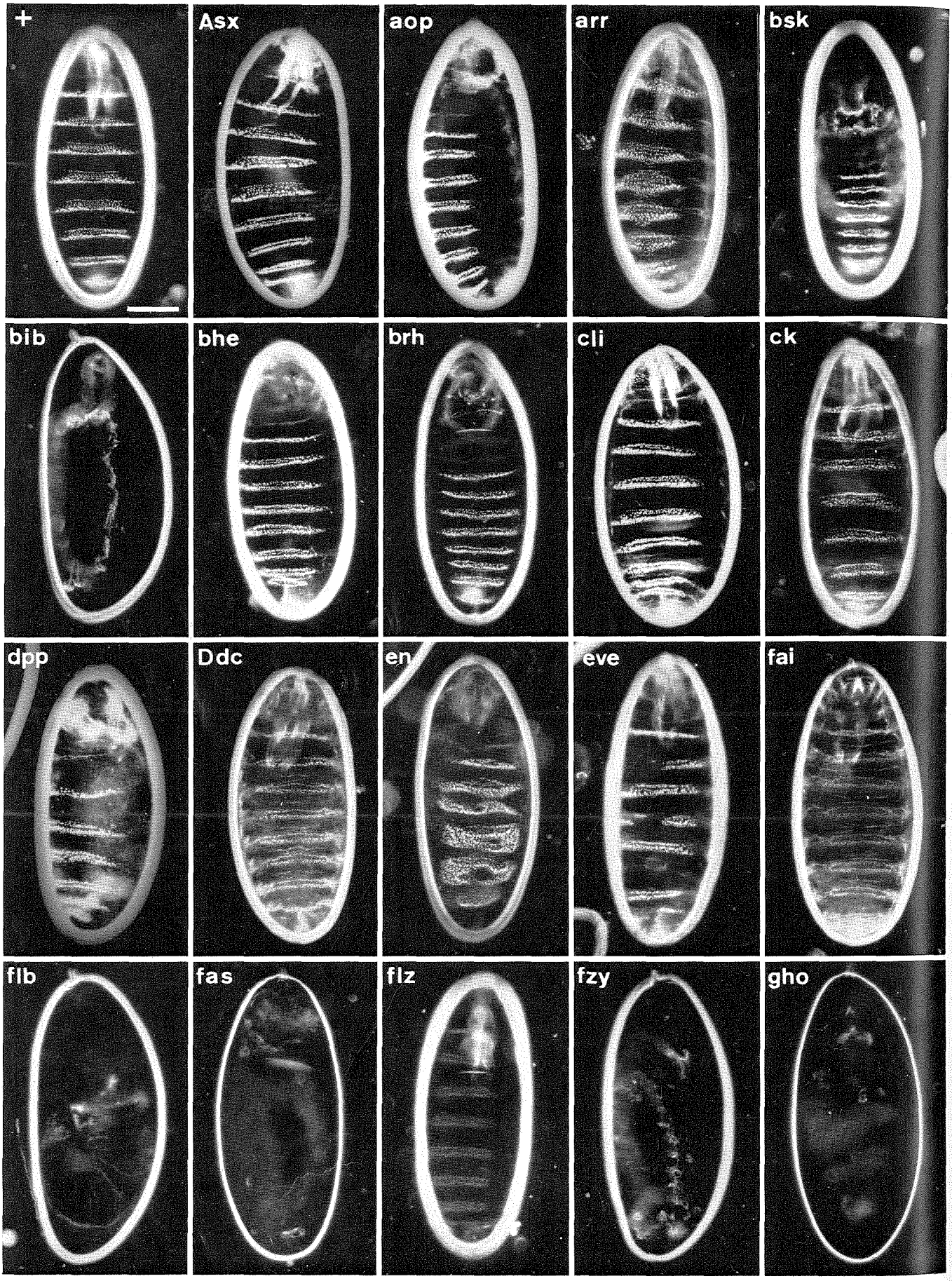
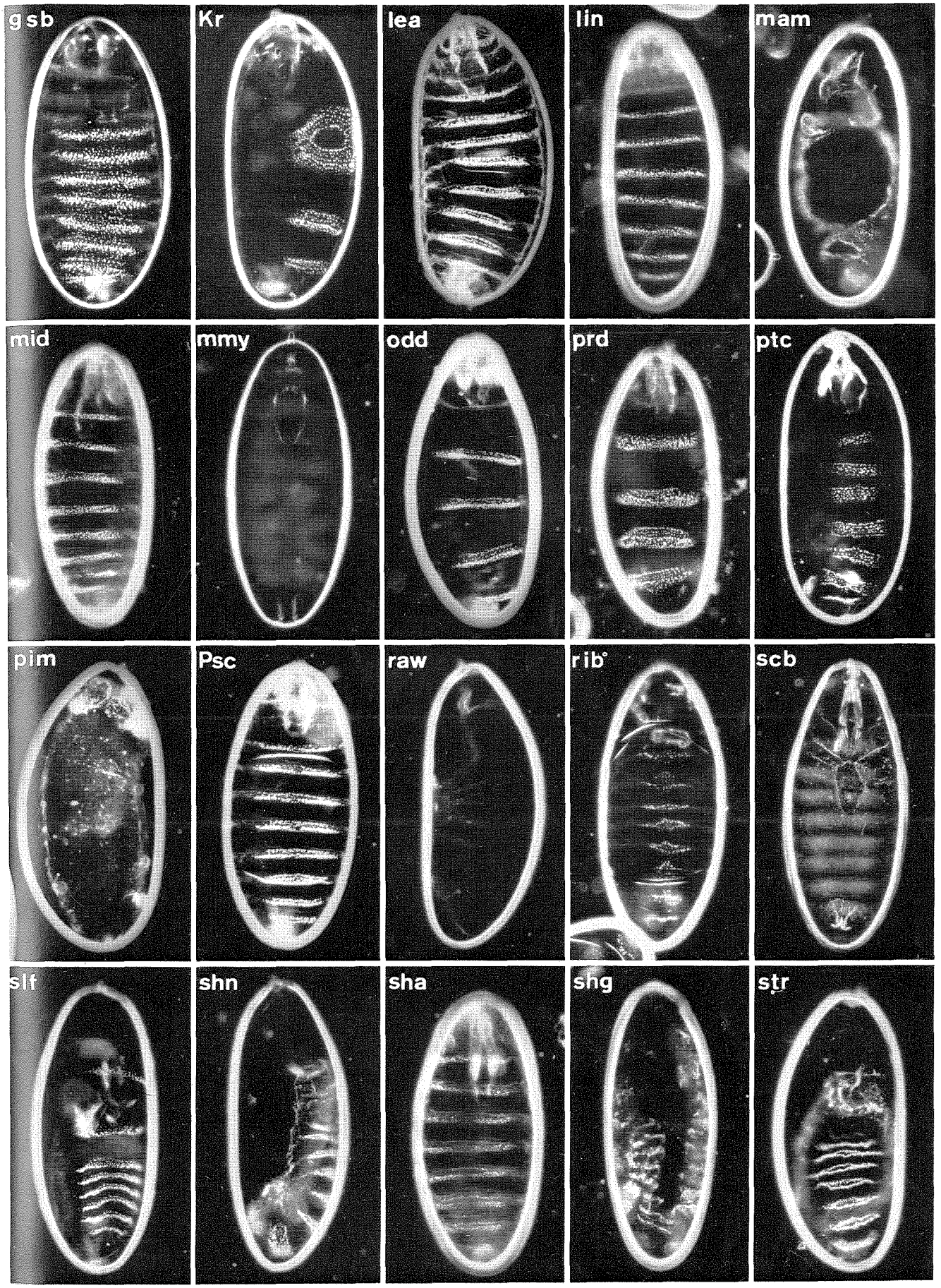


Fig. 4. Dark-field photographs of cuticle preparations of homozygous mutant embryos. A normal embryo, for comparison, is shown in the first picture. The *bar* represents 0.1 mm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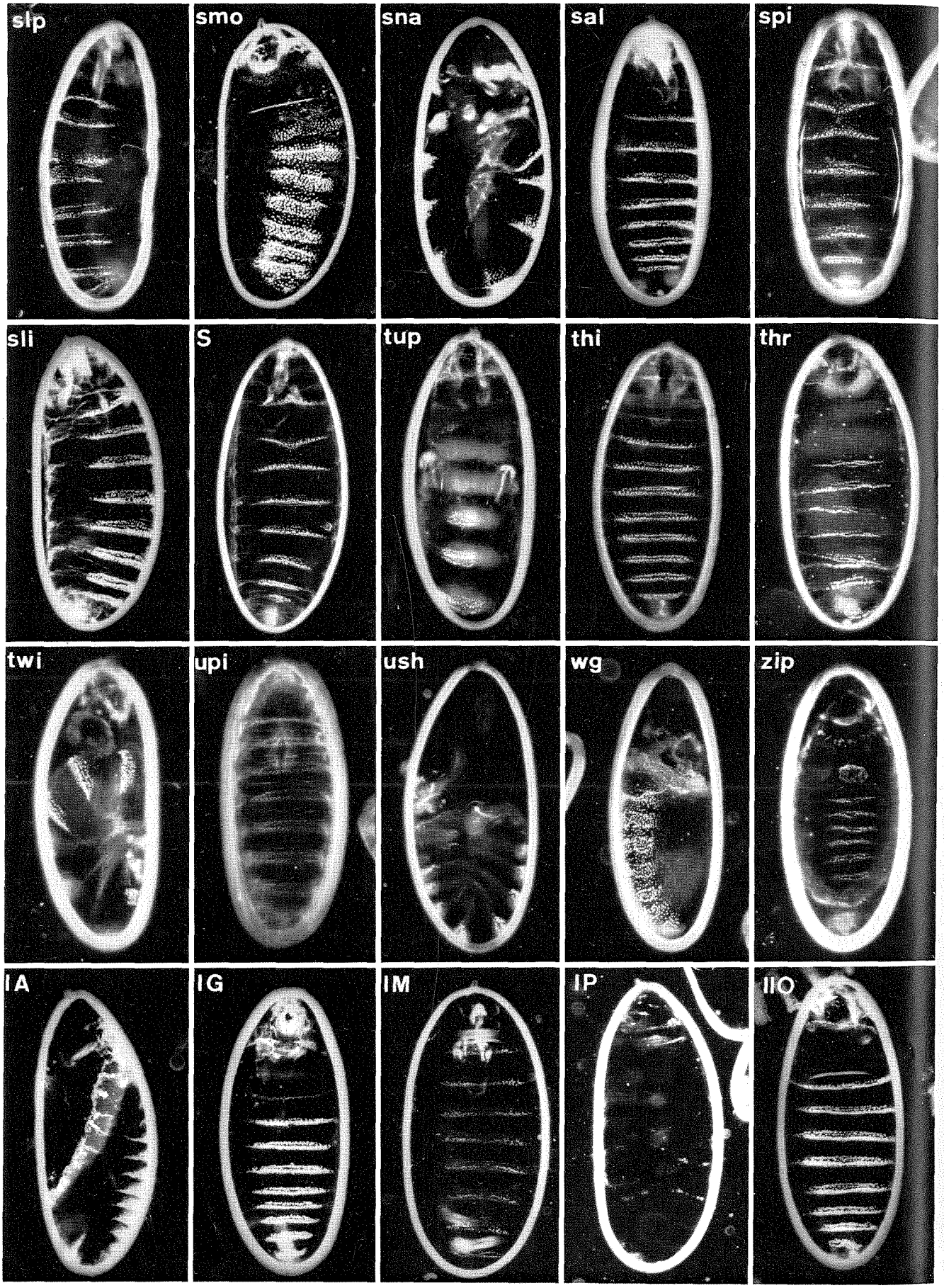
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Hypomorphic and temperature-sensitive alleles

As indicated in Table 3, in several complementation groups some alleles show a weaker phenotype than others. It appears likely that the strong phenotypes reflect the amorphic conditions of the respective genes, while the weaker phenotypes correspond to hypomorphic mutations. In several loci, temperature-sensitive (ts) alleles were found, which showed a weak or insignificant phenotype at low temperature and a strong phenotype at high temperature. Our screen has not been especially designed to isolate ts mutants, so our sample of 10 ts mutants per 272 mutants is probably under-represented.

In 13 instances it was possible to compare the true amorphic phenotype as represented by embryos homozygous for deficiencies of the respective loci with that of our alleles. In 9 of these cases, the strong alleles have the same phenotype as the respective deficiencies. For four loci all alleles have phenotypes weaker than the deficiency. In other cases, the homozygous deficiency phenotype was too poorly developed to discern differentiated structures (probably due to an additive effect of several lethal loci uncovered by the deficiency or a semi-dominant maternal effect associated with some of them (Garcia Bellido and Moscoso del Prado 1979). In these cases, transheterozygotes and strong alleles showed the same phenotype as the strong allele in the homozygous condition. Table 3 indicates the frequencies of the various types of alleles for all the loci.

Discussion

Phenotypic criteria

The work presented in the present paper is part of a programme aimed at identifying the genes involved in embryonic pattern formation in *D. melanogaster*. The differentiated embryo (larva) is a highly complex organism with many different cell types. In our screen for embryonic mutants we restricted ourselves to the analysis of the pattern of one particular tissue. We chose the larval epidermis, since the cuticle it produces is richly endowed with landmarks indicating position and polarity. The larval epidermis reveals perhaps the most striking aspect of embryonic pattern formation, segmentation, in greatest detail. The epidermis is derived from a large continuous region of the fate map and at hatching covers the entire larval body from anterior to posterior (Szabad et al. 1979; Lohs-Schardin et al. 1979). Thus it seemed likely that mutant effects on primarily soft parts of the embryo might also be detectable by a distortion or abnormal arrangement of the larval epidermis.

On the basis of a distinct phenotype visible in cuticular preparations, we have isolated 272 mutants mapping at 61 loci on the second chromosome. The mutant phenotypes cover a wide range of abnormalities. While the vast majority of the loci mutate to embryonic lethality, mutants at two of the loci, affecting the shape of denticles and hairs in the larva (*shavenoid* and *crinkled*) may survive to adulthood and in adults affect trichomes, and trichomes and bristles. Alleles at the *faint* locus are larval lethals. All other mutants are embryonic lethal and, in general, show a more severe deviation from normal than a mere morphological change in cuticle specializations. In many of the mutants internal organs are also strongly affected. From the analysis of the cuticular phenotype only, it often cannot be determined whether the effect of the mutant gene on the epidermal pattern is a secondary effect of more general distortions. The mutant phenotypes are briefly described in Table 3 and illustrated in Fig. 4.

Many of the genes identified on the basis of their cuticular phenotype are not likely to be directly involved in embryonic pattern formation, but rather in final differentiation of embryonic primordia. On the other hand, we believe that the majority of genes affecting embryonic pattern formation should show a phenotype visible in cuticular preparations and thus (given the degree of saturation reached in our screen, see below) should be contained in our collection. The mutants we would not have detected in our screen are, on the one hand, those in which internal organs are missing or abnormally shaped, while the cuticular pattern is normal, and, on the other hand, those which do not develop to a stage advanced enough to show any signs of cuticular differentiation. The first type of mutants would have been scored "normal" or poorly differentiated, the second mistaken for unfertilized eggs and not identified as embryonic lethal.

A large number of the "normals" from our screen have been inspected for abnormalities in the central nervous system (CNS) of living embryos using Nomarski optics, and, apart from a few cases with an apparent failure in the condensation of the nervous system, no other phenotype could be detected (C.M. Bate, personal communication). A further sample of mutants listed in Table 2 under "subtle phenotypes" were scored likewise for CNS defects using fuchsin-stained whole mounts. One mutant was found that showed massive cell death in the CNS, similar to the phenotype of homozygous *scute* deficiencies (J. Campos-Ortega, personal communication). Apart from the CNS, however, no other internal organs have been carefully scored.

In order to assess the frequency of mutants of the second class – the ones stopping development before the appearance of cuticular structures – we scored a large number of lethal lines (corresponding to approximately 2000 lethal hits) for early phenotypes in living embryos and did not find any. An independent set of data is based on the phenotypes of embryos homozygous for chromosomal deficiencies. The set of deficiencies on the second chromosome covers about 30% of the bands and all but one ($Df(2)vg^{B}$) show a phenotype which we would have detected in our screen or scored as normal or poorly differentiated. In cuticular preparations, vg^{B} embryos are indistinguishable from unfertilized embryos, whereas all other deficiencies tested, even very large ones, show at least signs of differentiation and many develop a normal cuticular pattern (Table 1).

The degree of saturation for embryonic visible phenotypes

Several lines of argument indicate that we have identified most of the loci on the second chromosome that mutate to an embryonic visible phenotype.

Rate of discovery of new loci. A simple calculation is based on the rate of isolation of mutants at new loci per chromosomes scored. Figure 5 shows that after scroing the first 25% of all lines, at least one mutant in more than 50% of the finally identified loci had been found, while in the last 25% of the lines only three new loci (5%) were found. The frequency of total mutants per lines scored was con-

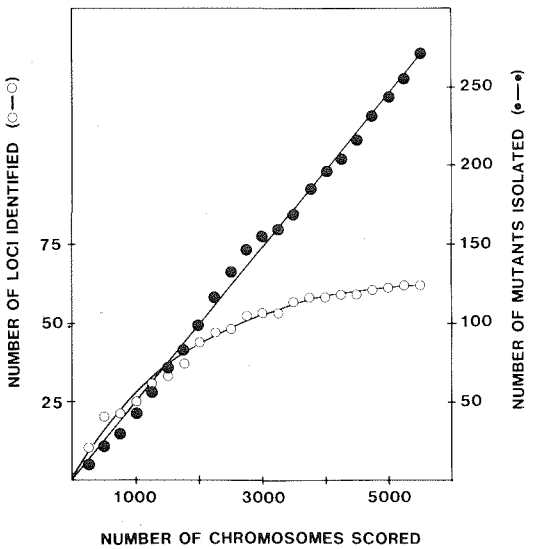

Fig. 5. The frequency of discovery of new loci compared to the total number of mutants isolated during the screen

stant throughout the screen. Thus, at the end of the screen alleles of previously isolated mutants were predominantly found. In a subsequent screen for third-chromosomal lethals (Jürgens et al. 1984), which also allowed the detection of second-chromosomal mutations with striking phenotypes (predominantly segmentation mutations), we found only alleles of previously identified loci.

Allele frequencies. Of the 61 loci, 79% are represented by more than one allele, the average being 4.5 alleles per locus. We assume that the majority of the mutations in loci represented by more than one allele are amorphic or hypomorphic mutations. Among the mutations in the 13 loci represented by only one allele, on the other hand, several might be antimorphic or neomorphic mutations in genes where amorphic mutations have no significant phenotype or are not embryonic lethal. Furthermore, the single allele class may also include other rare events like double mutations, neither of which produces a significant phenotype by itself. Our recombination experiments allowed the detection of synthetic phenotypes only if the individual mutants mapped rather far apart, and of the 17 initial single mutants 4 were found to be synthetic. Another possibility is that some single mutants are EMS-induced deficiencies and the phenotypes are caused by a cumulative effect of several genes of the poorly differentiated class. Therefore, estimations of the zero class based on the frequency of loci represented by one allele must be viewed with caution, since the one allele group is probably artificially enlarged.

The distribution of allele frequencies per locus (Fig. 2) is not random. Some loci appear to be mutable in much higher frequencies than others, the highest allele frequency exceeding the average by a factor of 4. On the other hand, allele frequencies might be severely biased in certain cases by the detectability of the phenotype in weak alleles. For instance, if an amorphic mutation in a certain locus leads to only a slight deviation from normal, a hypomorphic mutation in the same gene might not show a significant phenotype or might not even be embryonic lethal and thus go undetected. Similarly, loci with very strong extreme phenotypes may be detectable in comparatively weak alleles. The five 'hot spots' (*ptc, zip, flb, shn* and *shg*) all show very strong extreme phenotypes and are all represented by a number of weak alleles. The low allele frequency of most of the loci which show head defects as the major phenotypic feature might be explained by a similar argument. Head involution is a particularly sensitive process in embryogenesis in that slight disturbances lead to gross distortions, while even slighter ones may allow normal development.

While our screening criteria might have discriminated against weak alleles in several cases, it is possible that in other cases we have discriminated against strong alleles, if these show haplo-insufficient lethality or semi-lethality. It is striking that of the four loci for which we could show by comparison with the deficiency phenotype that our screen produced only weak alleles (*eve, Kr, prd* and *IG76*), three show semi-dominant lethality in hemizygous condition. *eve* is probably haplo-insufficient semi-lethal (Nüsslein-Volhard, unpublished work), *Kr* has a semi-dominant larval and adult phenotype (Gloor 1950; Wieschaus et al. 1984b) and deficiencies uncovering *IG76* have a semi-dominant maternal effect. These data indicate that in our screen we have discriminated in particular cases against certain types of alleles (depending on the locus; amorphic or hypomorphic). Thus the average allele frequency obtained is probably lower than the true hit frequency per locus.

In addition to the different level of detectability of different mutant phenotypes and probability considerations, the allele frequencies are likely to reflect the mutability of some of the genes with EMS. For 5 of the 13 loci represented by one allele in our screen, pains have been taken by others and by us to isolate more alleles. While the loci *sna, slp* and *Asx* (Ashburner et al. 1982; Simpson, personal communication; Jürgens et al. 1984; Jürgens, personal communication) seem to be mutable at a normal rate, EMS induced *dpp* alleles are very rare (Spencer et al. 1982), probably reflecting the haplo-insufficient lethality of the locus. The single *gsb* allele isolated in our screen turned out to be a small deletion (*Df(2R)IIX62*). In an EMS screen for *gsb* and *Kr* alleles, Preiss and Jäckle (personal communication) tested close to 20000 lethal hits and recovered 10 *Kr* alleles, one further *gsb* deletion but no single *gsb* point mutant. On the other hand, since all other significant deficiency phenotypes are accounted for by point mutants isolated in our screen, and this holds also for deficiencies on the third and first chromosome (Jürgens et al. 1984; Wieschaus et al. 1984a), non-mutability with EMS seems to be the exception rather than the rule.

Comparison with deficiency phenotypes. For the second chromosome, deficiencies covering about 30% of the bands have been tested for their homozygous lethal phenotype (Table 1). All significant embryonic visible phenotypes revealed in deficiency homozygous embryos have been accounted for by point mutants uncovered by the deficiencies, with the exception of *gsb* mentioned above. Of the 61 loci, 17 could be cytologically mapped using the available deficiencies which had been isolated by criteria other than an embryonic visible phenotype. This number (29%) corresponds well with the 30% of the bands uncovered by these deficiencies.

Calculation of the total number of lethal loci on the second chromosome. Given the saturation of the second chromosome for one particular phenotype, the total number of

lethal genes on the second chromosome may be calculated. The frequency of chromosomes having received at least one lethal hit in our experiment was 73% (4217/5764). Assuming a Poisson distribution of lethal hits, we can calculate the total number of lethal hits induced using the formula $P(0)=e^{-}\mu$ whereby $P(0)$ is the fraction of chromosomes without a lethal hit (0.27) and μ the average number of hits per chromosome. The calculation yields a total number of about 7600 lethal hits, 272 of which (3.6%) yielded an embryonic visible phenotype defining 61 loci. Assuming equal mutability of all lethal loci, these 61 loci should correspond to 3.6% of all lethal loci on the second chromosome. This calculation yields a total number of about 1700 lethal loci on the second chromosome, which is in fair agreement with estimates based on the number of bands (Lefevre 1974; Garcia-Bellido and Ripoll 1978).

Conclusions

In this paper we have described the isolation of mutants at 61 loci on the second chromosome of *D. melanogaster*. Some of the loci were known previously but most of them are new. All of them affect the formation of a normal cuticular pattern during embryogenesis. Although we cannot claim to have reached saturation for such loci in our screen, our calculations presented above indicate that our collection represents the great majority of such loci especially of those which mutate to a striking and unique embryonic visible phenotype. The number of genes which mutate to an embryonic visible phenotype is surprisingly small. For the second chromosome, these genes represent about 3% of all loci (approximately 2000) and similar numbers are obtained for the third and first chromosome. This means that the expression of the vast majority of the genes during embryogenesis is dispensible for the formation of a morphologically normal larva.

It is not the purpose of the present paper to describe the mutant phenotypes. Several of the phenotypes have been studied in some detail: those of the loci involved in segmentation (Nüsslein-Volhard and Wieschaus 1980; Nüsslein-Volhard et al. 1982), *Krüppel* (Gloor 1950; Wieschaus et al. 1984b) *engrailed* (Kornberg 1981), those of the loci of early neurogenesis *mam* and *bib* (Lehmann et al. 1983), and the phenotype common to the loci *twist* and *snail* (Frohnhöfer 1982; Simpson 1983) which, of all loci, shows the earliest visible deviation from normal, the lack of the ventral furrow during gastrulation. A superficial inspection of the pictures of mutant embryos in Fig. 4 shows that the degree to which the mutant pattern deviates from the normal pattern is great in some and barely detectable in others. From this picture it also becomes apparent that the genes described here are of very different importance for embryonic pattern formation. A comparison of the mutant phenotypes as shown in cuticular preparations further indicates that the majority of the genes have their own distinct phenotype. We estimate that the 61 loci of the second chromosome represent at least 41 distinguishable phenotypes and by a more detailed histological and developmental analysis of the mutant phenotypes this number may increase substantially. On the other hand, several of the mutant phenotypes are shared by loci mapping on the third or first chromosome (Jürgens et al. 1984; Wieschaus et al. 1984a). No clustering of genes with similar phenotypes is apparent; instead, the loci with embryonic visible phenotypes are spread more or less randomly throughout the genome.

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Notes added in proof

1) The locus *unpigmented* appears to be identical with the *Punch* locus (Lindsley and Grell 1968), based on complementation tests with Pu^2 and a dominant eye-colour phenotype of *upi* alleles

2) One representative allele of each locus can be obtained from the following address: Mid-America Drosophila Stock Center, Department of Biological Sciences, Bowling Green State University, Bowling Green, Ohio 43403, USA