Gpt, 87,000D (prob. dimer); Pgi, 97,000D (dimer).

The molecular weights of three enzymes were estimated directly from the gel filtration data by the method of Andrews (1965). Their elution profiles and position relative to the standards are shown in Figure 1a and 1b. Estimates of 51,000, 112,000 and 150,000 were obtained from Pgm (monomer), Idh (dimer), and fumarase (tetramer), respectively, using the least-squares regression from the five standards: $y = 5.257-0.002319x$. Fractions from this column were also assayed for activity of dipeptidase isozymes by starch gel electrophoresis. By this means it was possible to assign approximate molecular weights of 120,000 and 170,000 Daltons to the Dip-A and Dip-B isozymes, respectively (Voelker, Ohnishi and Langley 1979).

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Lindsley, D.E., L.S.B. Goldstein and L. Sandler. University of Washington, Seattle, Washington. Male sterility in maternal-effect mutants.

Seven mutations have been isolated in region 30-31 of chromosome 2 -- the so-called da-abo region. Of the five mutants that have been analyzed, all cause a maternal effect resulting in sex-specific embryonic lethality. In all cases, the severity of the maternal effect is sensitive to the heterochromatic constitution of the zygote, and the severity of the maternal effect is reduced if the experiments are carried out at 19° instead of 25°. The proposal was made that these five mutations define a cluster of functionally related genes (Sandler 1977).

We here report evidence that another of the seven mutations, mfs48, is also a member of the da-abo cluster. This evidence takes two forms: (1) mfs48 maps within the cluster, and (2) mfs48 exhibits phenotypic similarities to hup, one of the five known mutants in the cluster. On the basis of phenotype data presented here, we suggest a site of action for the genes in the da-abo cluster.

mfs48 was initially characterized as a male and female sterile with thin bristles; hup as a maternal-effect mutant which had held-up wings. hup was mapped to the right of da and tightly linked to abo; mfs48 was deficiency mapped to the left of abo and near da. We placed mfs48 to the right of da in the following way. Recombinant $J^{+}mfs48^{+}$ chromosomes from J da / mfs48 females were progeny tested for the da allele carried. Of 23 $J^{+}mfs48^{+}$ recombinants, 19 carried da and 3 da^{+}. Thus, the gene order is J-da-mfs48, with the da-mfs48 distance probably shorter than the J-da distance. This means that mfs48 is between da and abo and thus clearly maps within the cluster.

Both hup and mfs48 are lethal over a deficiency; we have found that in addition both are recessive semi-lethals. Thus, only 25% of mfs48 and 20% of hup homozygotes survive at 25°. We have also found that the fertility of mfs48 and hup homozygous males and females is temperature sensitive. At 23° both mutants are fertile (to some degree) in both sexes but at 28.5°, males and females are sterile (mfs48 females were only tested at 25° where they were found to be sterile). We have examined spermatogenesis in mfs48 and hup males raised at 23° and 28.5°. Males raised at 23° rarely show visible abnormalities in spermatogenesis. The testes are full of cells and motile sperm are observed in large numbers in the seminal vesicle. Males raised at 28.5° have no motile sperm and show a variety of defects during the later stages of spermiogenesis. The earliest defect which we have found is at the stage just after meiosis, the clew stage (Tates 1971). Cells at this stage and later contain micronuclei as well as macronuclei (see Fig. 1). In addition we find occasional spermatid nuclei with two basal bodies

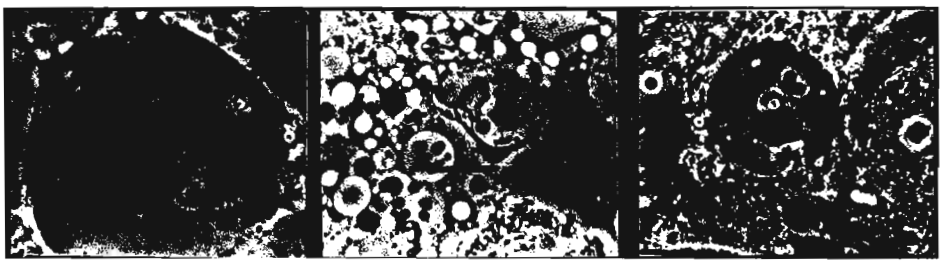

Fig. 1 Fig. 2 Fig. 3

Fig. 1. Nuclei from 28.5° mfs48. Single arrow indicates micronucleus. Double arrow indicates nucleus with two basal bodies attached (one is barely in the plane of focus). Magnification bar represents 10 μm.

Fig. 2. A spermatid nucleus with two axonemes in mfs48 was followed until the cell lysed. Here, the free nucleus is distorted by the drag from the two axonemes as it moves across the preparation. Magnification bar represents 10 μm.

Fig. 3. Two basal bodies with two axonemes attached to one nucleus in 28.5° hup spermatid. A single nebenkern (arrow) is associated. Magnification bar represents 10 μm.

(and two associated axonemes) attached to them (see figures). This is in striking contrast to wild type raised at 28.5° where spermatids contain single nuclei of uniform size and each spermatid receives only a single centriole which attaches to the nucleus and functions as a basal body. As yet, we have found no visible defects at earlier stages.

These observations suggest that the primary lesion in spermatogenesis in mfs48 and hup raised at 28.5° may be in the system which is responsible for the segregation of centrioles during division. Micronuclei may be caused by the occurrence of abnormal meioses in nuclei where the spindle poles and centrioles are not properly positioned.

Our observations on female sterility have as yet been uninformative because the female germ line is refractory to light microscopic investigation. We think, though, that the sterility in females could be caused by a defect in the accumulation of centrioles in the presumptive oocyte (Mahowald and Strassheim 1970). Clearly, abnormalities in centriole behavior could also have somatic effects resulting in the observed semilethality of mfs48 and hup. These observations lead us to wonder whether the loci in the da-abo region are all involved in the control of centriole movement during the development of soma and germ line. We are now in the process of testing this proposition and extending the cytological analysis.

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Lewis, R.A., T.C. Kaufman and R.E. Denell*. Indiana University, Bloomington, Indiana and *Kansas State University, Manhattan, Kansas. Genetic analysis of the Antennapedia gene complex (ANT-C): mutant screen of proximal 3R, section 84B-D.

The apparent localization to section 84B of several homoeotic loci involved in the determination and differentiation of anterior structures has led us to postulate a possible correlation between this series of developmental lesions and the well described, more distal bithorax complex (BX-C) (Lewis 1979).

A mutant screen designed to genetically dissect this region has been conducted, employing a deficiency ($Df(3R)Antp^{Ns+R17}$) spanning section 84B1,2-D11,12 which was generated as a revertant of the dominant homoeotic lesion Nasobemia (Duncan and Kaufman 1975). A total of 3133 chromosomes were treated either with EMS (.0125M) (Lewis and Bacher 1968) or X-rays (4000R). Chromosomes bearing lethal or visible mutations in the region of interest were recovered and maintained in balanced stocks with In(3LR)TM3,Sb Ser.