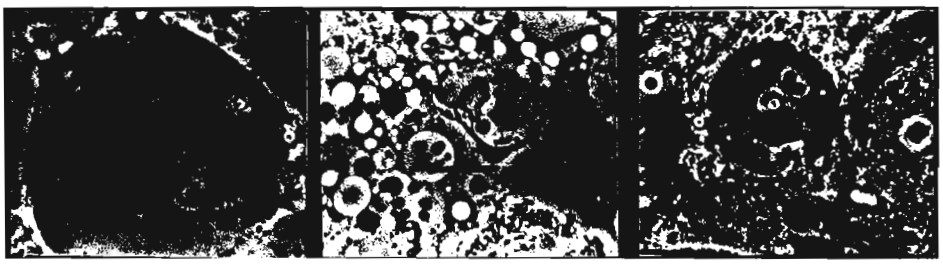

Fig. 1 Fig. 2 Fig. 3

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.

References: Mahowald and Strassheim 1970, J. Cell Biol. 45:306; Sandler 1977, Genetics 86:567; Tates 1971, Thesis 'S-Gravenhage: Drukkerij, J.H. Pasmans.

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.

Recovered mutations were named by the following alphanumeric code. A capital letter E or X indicates the inductive agent and is followed by a lower case letter designating the particular marked chromosome on which the lesion was induced: "a" (Dfd pP), "b" (Ki roe pP), "c" (Ki pP), "d" (pP cu) or "e" (Ki pP bx sr e^s). These two letters are followed by the initial of the discoverer and finally, a number to identify the particular mutation by its order of discovery.

85 lethal, 3 semi-lethal, and 6 visible mutations were recovered. Subsequent to balancing, each of these was crossed to four additional deficiencies (Df(3R)Scr, Df(3R)$Antp^{+R1P}$, Df(3R)-$Antp^{+R2}$, and Df(3R)dsx^{D+R2}). (For cytological limits see Duncan and Kaufman 1975; Kaufman 1978; and Fig. 1). These four deficiencies serve to subdivide the 84B-D interval into 7 regions and allow the identification of those mutations which reside in 84B1,2, the site of the Antennapedia complex (ANT-C) (Denell 1973; Duncan and Kaufman 1975). The results of this initial deficiency mapping are shown in Fig. 1.

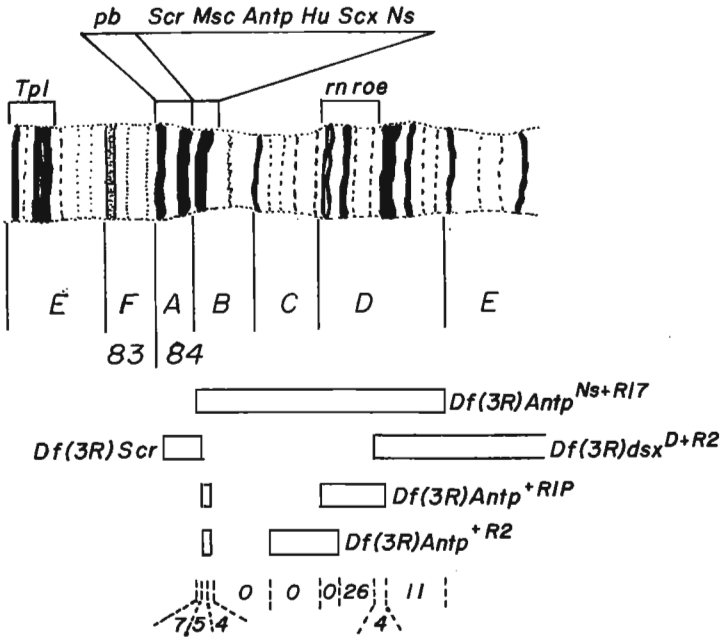

Fig. 1. Distribution of induced mutations obtained by deficiency mapping. (Tpl=Triplo-lethal; pb=proboscipedia; Scr=Reduced sex combs; Msc=Multiple sex comb; Antp=Antennapedia; Hu=Humeral; Scx=Extra sex comb; Ns=Nasobemia).

The most distal complementation group, defined by its lethal interaction with Df(3R)dsx^{D+R2}, consists of 11 members. Inter se crosses reveal four functional groups among the 11 mutations - two of two members each, one of one, and one of seven members.

Four mutations fail to complement Df(3R)dsx^{+R2} and Df(3R)$Antp^{+R1P}$. Inter se crosses define two functional groups, one of which is further divided into two subsites. This more complex group is characterized by a rotation of the male genitalia in surviving individuals.

26 mutations were recovered that fail to complement Df(3R)Ant^{+R1P}. A complementation analysis among all of these members reveals an extremely complex circular pattern.

37 mutations fail to complement only the original screening deficiency. The great majority of them have been shown by a preliminary recombination analysis to reside quite far from 84B-D. Several members of this group in various heterozygous combinations with one another, confer a marked swelling on the mesothoracic and metathoracic legs. They are currently being mapped more precisely.

The deficiency analysis established three complementation groups in the vicinity of the 84B1,2 doublet. The distal-most of these is defined by failure to complement Df(3R)$Antp^{+R1P}$ and Df(3R)$Antp^{+R2}$ and is comprised of four mutations. The proximally neighboring group fails to complement Df(3R)Scr as well as the above two deficiencies. There are five of this class of mutation. The most proximal collection fails to complement only Df(3R)Scr and contains seven lesions.

Inter se combinations within each of these deficiency defined regions have shown that the members of the distal-most group (EdR16, EdR17, EbR27, XbD2) form a single complementation group. The middle group (EcR10, EbD7, EcK5, XbK4, EeR4) also forms a single unit. Finally, combination of members of the most proximal group reveal two separate complementation units. One (EdK6, EdD8, EdR18) is further characterized by a dominant reduction in the number of sex comb teeth similar to that seen in male heterozygotes of either Df(3R)Scr or Df(3R)$Antp^{Ns+R17}$. The other complementation unit (EbR11, XaK2, XaK5, XaK26) is complex and is shown in Fig. 2.

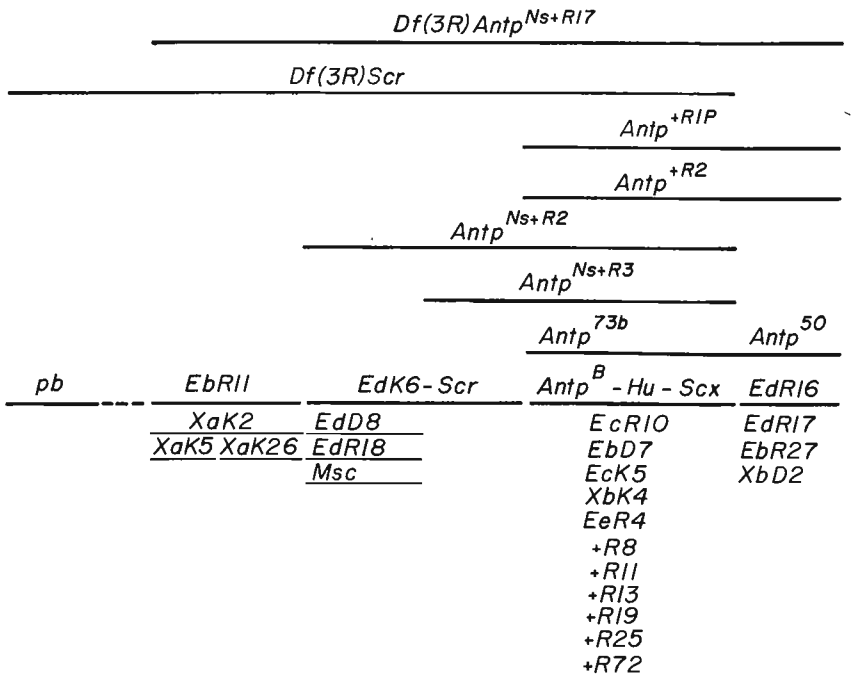

Fig. 2. Complementation map of induced mutations within Antennapedia complex. Localizations determined by complementation patterns of new mutations with deficiencies and extant homoeotic lesions resident in the 84B1,2 doublet. (Note: +R8, +R11, etc., refer to revertants of $Antp^{Ns}$.)

Crosses between each of these 16 lethal lesions and the homoeotic lesions known to map to 84B1,2 were performed to further delineate these sites. In this manner, the group showing the reduced sex comb phenotype was shown to be allelic to Multiple Sex Comb (Msc), which also displays this phenotype. The next most distal group was similarly shown to be allelic to Antennapedia (Antp), Extra Sex Comb (Scx) and Humeral (Hu). Further, the members of the most distal site failed to complement the recessive lethality of $Antp^{73b}$ but were viable in combination with $Antp^{B}$ and Scx. The remaining group to date has not been associated with any known homoeotic lesion.

A clarification of the proximal distal orientation of these four groups has been provided by crossing the 16 lethal lesions to 8 additional revertants of $Antp^{Ns}$ (Duncan and Kaufman 1975; Denell 1973). The results of these crosses confirm the proximal-distal array found in the deletion analysis and further demonstrate that the EbR11 group is proximal to the other three groups (Fig. 2).

A most interesting addition to the list of homoeotic transformations attributable to lesions in 84B1,2 has been revealed by a mutation in the Sex Combs Reduced (Scr) group. As previously mentioned the number of sex comb teeth produced in males heterozygous for these lesions and the TM3 balancer ranges from 5 to 8 as compared to the normal 8 to 12. Rare surviving individuals of the genotypes EdR18/Df(3R)Scr and EdR18/$Df(3R)Antp^{Ns+R17}$ frequently possess no sex combs at all. Those teeth which are occasionally present are not rotated. Further aspects of the chaetotaxy of these legs (e.g., the absence of transverse rows on the tibia) indicate that the prothoracic leg is transformed into a mesothoracic leg. An additional transformation can also be seen in EdR18/Df(3R)Scr and in EdR18/$Df(3R)Antp^{Ns+R17}$ individuals. The first three rows of pseudotracheae of the labial palps are transformed into what appears to be maxillary palpus. We interpret this as indicating an interaction of the Scr locus with a site proximal to it and contained within the limits of the Df(3R)Scr lesions. The possibility that this site is the proboscipedia locus is currently being investigated.

References: Denell, R. 1973, Genetics 75:279; Duncan, I.W. and T.C. Kaufman 1975, Genetics 80:733; Kaufman, T.C. 1978, Genetics 90:579; Lewis, E.B. 1979, Nature 276:565; Lewis, E. B. and F. Bacher 1968, DIS 43:193.