prevent coagulation and freezing of blood samples at the −1.9°C seawater temperature. To calculate P_aN_2 we corrected the inert gas content for dilution. The latter was determined by marking the flush solution with tritiated water (seals 1 and 2) or calculating the hemoglobin ratio of the diluted sample to undiluted blood obtained simultaneously from the sampling line (seals 3 and 4).

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15. Supported by the NSF Division of Polar Programs grant 8100212, SFB Kardiologie 30 of the Deutsche Forschungsgemeinschaft, and Landesamt fur Forschung, Nordrhein-Westfalen, Duesseldorf. Studies were performed in accordance with NMFS permit No. 394. We thank the National Science Foundation, Antarctic Services, Inc., and the U.S. Navy for field assistance. We also thank F. Klocke and H. Rahn for their invaluable advice on measuring nitrogen tensions.

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Effects of Genomic Position on the Expression of Transduced Copies of the *white* Gene of *Drosophila*

Abstract. *The* white *gene of* Drosophila *is expressed normally when introduced at many different sites in the genome by P-element–mediated DNA transformation, but is expressed abnormally when inserted at two particular genomic positions. It is now demonstrated that the mutant expression in these two cases is caused by the surrounding chromosomal region into which the* white *gene has been inserted. The* white *gene could be moved from these two positions, where it confers a mutant phenotype, to other positions in the genome where it confers a wild-type phenotype. However, flies in which* white *has been moved to one new location have an unusual mosaic phenotype.*

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The relation between the chromosomal location of a gene and its expression has been studied in *Drosophila* by means of chromosomal rearrangements such as inversions and translocations. Rearrangements can result in the inactivation of genes located at some distance from the rearrangement breakpoints. This inactivation commonly occurs in only a portion of the cells in which the gene is normally expressed, causing the fly to have a mosaic phenotype. Such variegating position effects are usually associated with rearrangements that bring euchromatic genes into the proximity of centromeric heterochromatin (*1*).

The development of the technique of P-element–mediated DNA transformation in *Drosophila* has expanded our ability to study genomic position effects. A gene, carried within a P transposable element, can be introduced at many genomic positions, and its expression at these different positions can be compared (*2*). Several genes that have been transformed into the germline by this method show proper developmentally regulated expression at a variety of euchromatic positions (*2, 3*).

The *white* gene confers an essentially wild-type (red-eyed) phenotype (*4*) when transduced to any of at least 20 different chromosomal locations by P-element vectors (*5, 6*). In contrast, three cases have been reported in which a transduced *white* gene confers a mutant mosaic eye color (*5, 6*). Flies carrying one copy of the transduced *white* gene A^R4-3 are more darkly pigmented in the anterior portion of their eyes than in the posterior portion. This transduced gene is located near the centromeric heterochromatin of chromosome arm 2L. Another transformant, A4-4, has a transduced *white* gene at the end of the right arm of chromosome 3 and has a yellow eye color with scattered flecks of red (*5*). Gehring *et al.* (*6*) have reported a third mosaic transformant, $P(w^{var})$, that has red-orange variegated eyes, and a transduced *white* gene at the left end of chromosome 2.

We have suggested that the mutant phenotypes of A^R4-3 and A4-4 are the result of genomic position effects because we could not detect alterations of their transduced *white* DNA that might account for the mutant phenotypes (*7*). Furthermore, the *white* gene in the A^R4-3 transformant is located adjacent to centromeric heterochromatin, which is capable of eliciting variegating position effects (*1*). However, their mutant phenotypes might be due to mutations within the transduced genes that were not detectable by the DNA blotting methods used. To test this possibility we have moved the transduced *white* gene in A^R4-3 and A4-4 to new positions. Relocating the gene should result in a wild-type eye color at most new positions if its mutant phenotype is due to a position effect, but not if it is due to a mutation intrinsic to the gene.

To relocate the transduced *white* gene in A^R4-3 and A4-4, we mobilized the P-*white* transposon in these two strains (Fig. 1). The P-*white* transposon cannot catalyze its own transposition, but can be induced to transpose by injecting a nondefective P element. Embryos of each strain were injected with plasmid DNA containing the "wings-clipped" P element, which is a P element with a deletion in a portion of one of the terminal inverted repeats (*8*). The "wings-clipped" P element can induce the transposition of P-element transposons, but does not itself integrate into the chromosomes (*8*). Therefore, the P-*white* transposon should transpose in the germ cells of some of the injected embryos but should be stable in subsequent generations.

Progeny of the injected embryos having eye colors different from A^R4-3 and A4-4 were chosen for further analysis and are referred to as revertants. Seventeen independent revertants were recovered from the progeny of injected A^R4-3 embryos and five independent revertants were recovered from the progeny of the injected A4-4 embryos (*9*). Hybridization of *white* gene DNA to polytene chromosomes (*10*) resulted in the identification of 13 new chromosomal locations of the P-*white* transposon among the revertants of A^R4-3 and two new locations among the revertants of A4-4 (*11*). The sites to which the P-*white* transposon moved were distributed over the euchromatic portions of all of the major chromosome arms (Fig. 2). Restriction digest analyses (*12*) of the DNA of these revertants are consistent with there being a single intact *white* gene at each of these new sites. An example of a DNA blot hybridization which was part of this analysis is shown in Fig. 3. Flies in which the *white* gene from A^R4-3 has been inserted at the 24CD site (*13*) have a new mosaic pattern of pigmentation (described below). Flies having a mobilized *white* gene at any one of the other new sites had a wild-type eye color, as judged by visual inspection (*14*). We conclude that the mutant phenotypes of the parental transformants A^R4-3 and A4-4 are the result of genomic position effects, as the same copy of the *white* gene that confers a mutant phenotype in each of these parental transformants could confer a wild-type phenotype at other sites.

In addition to the revertants described above, the transduced *white* gene remained within the same chromosomal region as in the original mutant

transformant in six of the revertants of A^R4-3 and three of the revertants of A4-4, as determined by in situ hybridization of polytene chromosomes. None of the three A4-4 revertants in which the *white* gene remained in the original region had a completely wild-type eye color. Two of these had orange eyes with scattered darker spots, while the third had red eyes dotted with darker spots. Among the derivatives of the other original mutant, A^R4-3, four of the six revertants in which the transduced *white* gene remained at the base of chromosome arm 2L had a completely wild-type phenotype. The other two such derivatives had only a very small region of unpigmented tissue along the posterior border. Even though there was no apparent change in the chromosomal position of the P-*white* transposon in these A^R4-3 and A4-4 derivatives, DNA blot-hybridization analysis (*12*) indicates that in every case there were structural changes in the transposon or flanking DNA. Part of the analysis of the derivatives of A^R4-3 is shown in Fig. 3. We cannot deduce the nature of the changes that took place from these blots. The changes may include transpositions to nearby sites, changes within the transposon, or deletions or other rearrangements of the DNA adjacent to the transposon which are too small to be detected cytologically. Further analysis of these events may aid in defining the sequence elements within the flanking DNA that modify the expression of *white* in the original mutant transformants.

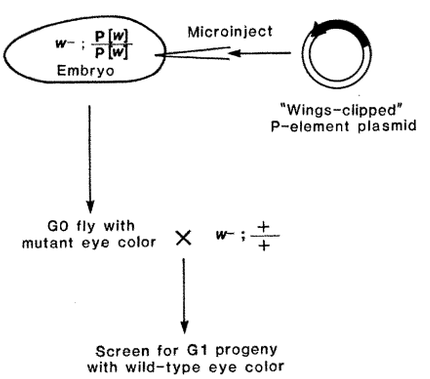
The mobilization of the P-*white* transposon of A^R4-3 also produced a derivative with a heritable, novel mosaic pattern of eye pigmentation (*15*). The transduced *white* gene of this derivative is located in the 24CD cytogenetic region (*13*) and we will refer to it as A^R4-24. The ventral halves of the eyes in the A^R4-24 flies are more darkly pigmented than the dorsal halves (Fig. 4C). This pattern is like that observed in A^R4-3 flies in that one part of the eye is reproducibly more darkly pigmented than the other. However, the eye pigmentation patterns of A^R4-24 and A^R4-3 are oriented perpendicular to each other.

A^R4-3 and A^R4-24 are fundamentally different from other *white* position effect mutants in which the locations of patches of pigmented and unpigmented tissue vary, more or less at random, in the eyes of flies carrying the same mutant allele. The reproducible patterns of pigmentation in A^R4-3 and A^R4-24 indicate that the phenotypic expression of *white*, at least in these mutants, must be sensitive to positional information in the developing eye (*16*).

A^R4-3 and A^R4-24 differ in the effect of gene dosage on the pigmentation pattern. Flies with two copies of A^R4-3 (Fig. 4B) have a larger portion of their eyes darkly pigmented than flies with one copy (Fig. 4A), and this portion has a more wild-type color. In flies with two doses of A^R4-24 (Fig. 4D), the area of darkly pigmented eye tissue remains nearly the same or may even be slightly less than in flies with one dose (Fig. 4C). The lightly pigmented dorsal half of the eye of flies with two doses of A^R4-24 is even more lightly pigmented than that of flies with one dose.

A^R4-3 and A^R4-24 are similar in the effect of the z^1 allele of the *zeste* (*z*) gene on the eye pigmentation pattern. The z^1 allele is recessive to z^+ in most cases in its interaction with normal or transduced *white* genes (*5, 6, 17*). In addition, two doses of a normal or transduced *white* gene are usually required in combination with z^1 to reduce the level of eye pigmentation (*5, 6, 18*). A^R4-3 and A^R4-24 are unusual in that the position of the pigment border shifts in heterozygous z^1w^-/z^+w^- females carrying one copy of the transduced *white* gene (*19*). In such A^R4-24 females, the pigment boundary is displaced toward the anterior-ventral corner (Fig. 4E), whereas in A^R4-3 it has moved in the direction of the anterior edge (*5*). In males that are hemizygous for z^1w^- and carry one copy of A^R4-24, only a small region of the eye in the anterior-ventral corner is pigmented (Fig. 4F) (*20*). Thus, the transduced *white* genes in A^R4-24 and A^R4-3 are hypersensitive to the z^1 allele, which has the effect of decreasing the area of pigmentation.

We believe that the mosaic mutant phenotype of A^R4-24 is probably the result of a genomic position effect. In initial experiments designed to induce further transpositions of A^R4-24, we have recovered flies with a wild-type eye color. The chromosomal region in which the *white* gene is inserted in A^R4-24 would not be expected to inactivate an inserted euchromatic gene (*21*). A transduced gene may be more sensitive to position effects than a gene in a chromo-



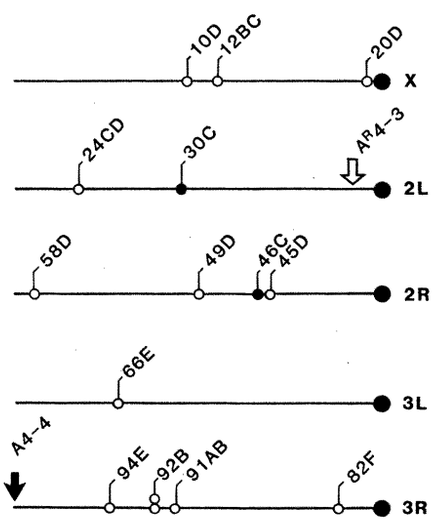


Fig. 1 (left). Strategy for recovering transpositions of the P[*w*] transposons A^R4-3 and A4-4. DNA (50 μg/ml) of the plasmid pπ25.7wc ("wings-clipped") (*8*) was injected into embryos (*23*) homozygous or hemizygous for w^{1118} and homozygous for the transposon P[(*w,ry*)A^R]4-3 or P[(*w,ry*)A]4-4. (The notation P[(*w,ry*)A] indicates one of a series of P-element transposon constructs carrying the *white* and *rosy* genes; the 4-3 and 4-4 suffixes refer to fly strains with this transposon inserted at particular sites.) The injected plasmid DNA causes the destabilization of the transposon in some of the cells of the germline of the injected embryo. The G0 flies that grew from the injected embryos all had the mutant eye color of their parents. These G0 flies were then mated to w^{1118} flies [this allele contains a partial deletion of the *white* gene and these flies have bleached white eyes (*5*)]. The plus in the diagram denotes a normal autosome without a transposon. The resulting G1 progeny carried a single copy of the original P[*w*] transposon and therefore had the parental mutant eye color, unless a transposition or other genomic rearrangement induced by the P element had taken place in the germline of the G0. For each revertant the chromosome (or chromosomes) bearing the *white* gene that confers the revertant eye color was identified by sex linkage for insertions on the X chromosome, or by its segregation from dominantly marked autosomes. When the transposon had moved to a new chromosome, it was genetically isolated from the chromosome bearing the original A^R4-3 or A4-4 gene. Fig. 2 (right). New sites of P-*white* insertions in derivatives of A^R4-3 and A4-4. The euchromatic portions of the five major chromosome arms are represented by horizontal lines, oriented with their centromeres (large closed circles) to the right. The original locations of the A^R4-3 and A4-4 transposons are indicated by the open and closed heavy arrows, respectively. New sites of P-*white* transposons are marked along the lines with open circles for derivatives of A^R4-3 and with closed circles for derivatives of A4-4. Above each circle is noted the cytogenetic region, according to the system of Bridges (*24*). Two independent insertions occurred in the 92B region in revertants of A^R4-3, but these were found by DNA blot analysis (*12*) to have occurred at different sites or be in opposite orientations (lanes 3 and 4 of the left panel of Fig. 3).

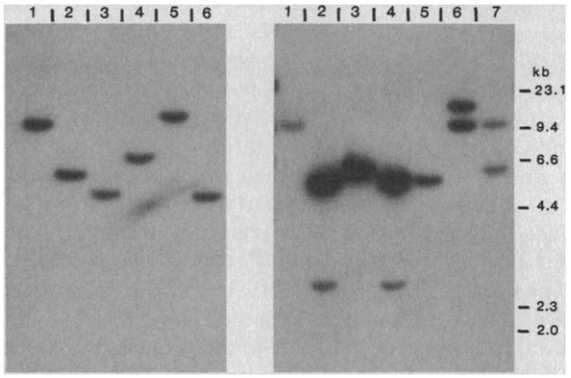


Fig. 3. DNA blot hybridization analysis of derivatives of A^R4-3 (*5*). DNA extracted from adult flies was digested with Bam HI, separated by electrophoresis on a 0.6 percent agarose gel, blotted onto nitrocellulose, and hybridized with a nick-translated plasmid DNA probe containing the Bam HI (+4.7)–Eco RI(+6.6) segment of *white*. This probe hybridized to the Bam HI fragment spanning the junction between one end of the transposon and the flanking DNA. All of the flies had the w^{1118} allele in place of the normal *white* gene; w^{1118} contains a deletion of part of the *white* gene including all of the DNA complementary to the probe used in this hybridization. Therefore, the only hybridization is to the transduced *white* gene. Lane 1 of both panels contains DNA from the parent line, A^R4-3. Lanes 2 to 6 of the left panel contain the DNA's of revertants of A^R4-3 in which P-*white* is known to have transposed to new sites on the basis of in situ hybridizations to polytene chromosomes. Lanes 2 to 7 of the right panel contain the DNA's of revertants of A^R4-3 in which, by in situ hybridization, the transposon appears to remain at its original position. The more intense hybridization in lanes 2 to 4 reflect a greater amount of DNA inadvertently loaded in these lanes. The distance of migration of markers of a Hind III digest of phage λ DNA are indicated at the far right.

somal rearrangement, because of the small size of the transduced segment (*22*).

In conclusion, this study establishes the utility of P-element–mediated DNA transformation for the creation and analysis of position effect mutants. We have shown that the mutant expression of a transduced *white* gene at each of two chromosomal locations is due to a position effect; the wild-type expression of the transduced *white* gene was restored after it was induced to transpose to new locations. We have also recovered phenotypic revertants in which nearby transpositions or local rearrangements of flanking sequences have occurred. Injection of the "wings-clipped" P element is an efficient method for inducing such transpositions and rearrangements; these alterations were recovered approximately ten times more frequently than the original insertions of the plasmid containing the P-*white* transposon when it was coinjected with a P-element helper (*5*). Furthermore, when the P-*white* transposon was induced to transpose, it moved to a wide variety of new sites scattered throughout the euchromatin, indicating that P elements are not restricted during transposition to nearby chromosomal sites. The presence of *white* at one of these new sites, at 24CD, results in a novel mosaic eye pigment pattern, suggesting that expression of the *white* gene at this site is sensitive to cell position in the eye.

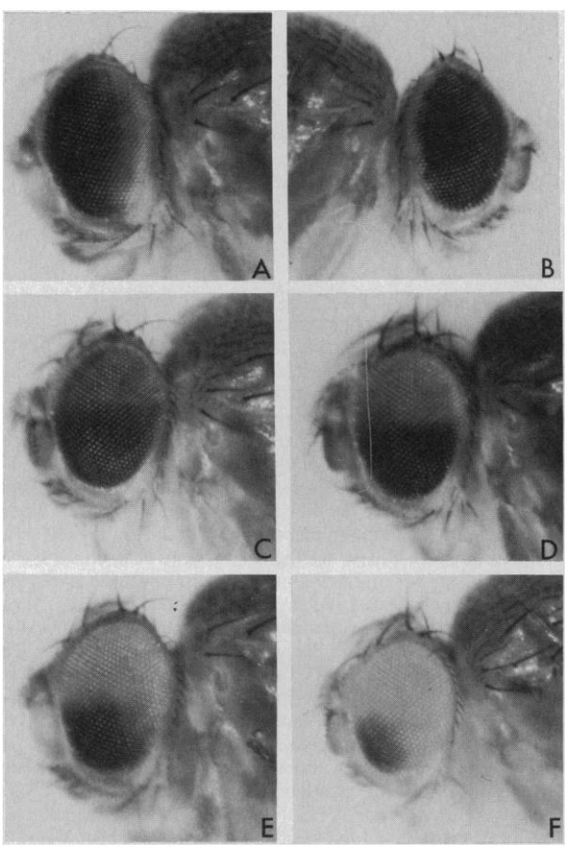


Fig. 4. Mosaic pigment patterns. An eye of a fly of the following genotypes is shown: (A) w^{1118}/w^{1118}; P[(*w*,*ry*)A^R]4-3/+; (B) w^{1118}/w^{1118}; P[(*w*,*ry*)-A^R]4-3/P[(*w*,*ry*)A^R]4-3; (C) w^{1118}/w^{1118}; P[(*w*/*ry*)A^R]4-24/+; (D) w^{1118}/w^{1118}; P[(*w*,*ry*)A^R]-4-24/P[(*w*,*ry*)A^R]4 - 24; (E) $z^1w^{1118}cv/w^{1118}$; P[(*w*,*ry*)A^R]4-24/+; (F) $z^1w^{1118}cv$/Y; P[(*w*,*ry*)-A^R]4-24/+. Each fly was grown at 25°C and was 5 days old before the photograph was taken. Dorsal is toward the top in all photographs and anterior to the left in all except panel B where anterior is to the right.

References and Notes

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4. The *white* gene product is required for the deposition of pigments in the eye, as well as in a number of other tissues; in null mutants the eyes and these other tissues are white. For a review of *white*, see B. H. Judd, in *The Genetics and Biology of* Drosophila, M. Ashburner and E. Novitski, Eds. (Academic Press, London, 1976), vol. 1b, p. 767.
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7. The A^R4-3 transposon contains a rearrangement 3′ to the transcribed region of the *white* gene. For details of the sequences rearranged and experimental evidence that this rearrangement is not directly responsible for the mutant phenotype, see (*5*).
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9. The five A4-4 revertants and 17 A^R4-3 revertants arose in the G1 progeny of 32 and 41 fertile injected flies, respectively. White-eyed G1 progeny were also observed. We assume that these descended from the germ cells of G0 flies in which the P-*white* transposon was partially or completely excised. Both types of events are induced by P elements [(*8*); K. O'Hare and G. M. Rubin, *Cell* **34**, 25 (1983); R. A. Voelker *et al.*, *Genetics* **107**, 279 (1984); S. B. Daniels *et al.*, *ibid.* **109**, 95 (1985)].
10. Males of each revertant stock were mated to w^{1118} females; polytene chromosome squashes were prepared from the F1 larvae by a method devised by J. Lim of the University of Wisconsin at Eau Claire (unpublished). These were hybridized with a ^{3}H-labeled complementary RNA *white* probe (*5*). The sites of hybridization were scored by referring to the photographs of G. Lefevre, in *The Genetics and Biology of* Drosophila, M. Ashburner and E. Novitski, Eds. (Academic Press, New York, 1976), vol. 1a, p. 31.
11. The 13 new sites were found in 11 revertants of A^R4-3; two of the revertants carried the transduced *white* at two new sites, 45D+94E and 12BC+92B.
12. The preparation of genomic DNA and the hybridization of DNA filter blots were performed as described (*5*). Three DNA blots were done for each revertant. The first blot (*5*) showed the number of sites at which the *white* DNA was inserted. The other two blots were tests for whether or not the transduced *white* DNA remained intact. For revertants of A4-4, genomic DNA digested with a combination of Xba I and Eco RI was probed on one blot with both the Bam HI(+1.4)–Hind III (+3.3) and Hind III (+3.3)–Bam HI(+4.7) *white* DNA fragments (where the numbers in parentheses refer to map positions in kilobases relative to the point of

insertion of the copia element in the *white-apricot* allele; for a map of *white*, see R. Levis, P. M. Bingham, G. M. Rubin, *Proc. Natl. Acad. Sci. U.S.A.* **79**, 564 (1982); for a map of the transposons, see (*5*). This probe hybridizes to a 5.9-kilobase (kb) fragment in this digest representing the 5′ half of the *white* DNA of the transposon. The w^{1118} allele does not hybridize with this probe. An identical DNA blot was hybridized with a probe containing both the Sal I(-2.7)–Sal I(-1.3) and Sal I(-1.3)–Sal I(−0.5) segments. This probe hybridizes to a 5.8-kb fragment in this digest representing the 3′ half of *white* in the transposon plus a 6.2-kb fragment from w^{1118}. For revertants of A^R4-3, genomic DNA digested with Xba I and Eco RI was hybridized with a probe containing the Xba I (+0.7)–Eco RI (+6.6) *white* segment. This segment hybridizes to the 5.9-kb fragment representing the 5′ half of the *white* DNA in the transposon. A Hind III digest of A^R4-3 revertants was hybridized with the Bam HI(+1.4)–Hind III(+3.3) *white* segment that hybridizes to the 6.2-kb band. This band represents the 3′ half of *white* in the A^R4-3 transposon and does not hybridize to w^{1118} DNA.

. The designation 24CD refers to a polytene chromosome region in the maps of C. B. Bridges (*24*).

. Both cytogenetic and blot analysis indicate that the three wild-type revertants of A^R4-3 having new sites of *white* on the second chromosome also retain the transposon at the original site (near the centromeric heterochromatin of chromosome arm 2L). This does not prove that transposition of P-transposons is replicative, however, since it might have been the copy of A^R4-3 on the other homolog or the sister chromatid of the injected embryo which transposed. For these three revertants, we have not constructed strains that carry *white* at these new sites alone. Therefore, we can only infer that the wild-type phenotype of each of these revertants was caused by the *white* gene at the new site.

. The chromosome containing this derivative also carried the parental A^R4-3 transposon. The presence of this chromosome resulted in a nearly wild-type eye color. The original and new sites were subsequently separated by recombination to reveal the novel mosaic phenotype conferred by the gene at the new site.

. Certain features of eye development demonstrate the existence of positional information in the eye. An equator of symmetry in photoreceptor arrangement bisects the eye into dorsal and ventral halves. A preferential boundary for cell clones is located parallel to the equator but does not always coincide with it [H. J. Becker, *Z. Indukt. Vererb. Lehre* **88**, 333 (1957); W. K. Baker, *Dev. Biol.* **62**, 447 (1978); D. F. Ready, T. E. Hanson, S. Benzer, *ibid.* **53**, 217 (1976); J. A. Campos-Ortega and M. Waitz, *Wilhelm Roux Arch. Entwicklungsmech. Org.* **184**, 155 (1978)]. We do not know the precise position of the differential pigment border in the eyes of A^R4-24 flies (Fig. 4C) relative to these other two boundaries or whether there is a relation between them. It is also possible that there is a relation between the pigment pattern in the eyes of A^R4-3 flies (Fig. 4A) and the morphogenetic furrow, which moves from the posterior to the anterior across the eye-antennal imaginal disk of the third-instar larva.

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The 11 wild-type revertants with new insertions of *white* on autosomes have been tested for the interaction of a single copy of the P-*white* transposon with z^1. Males of the genotype $z^1w^{1118}cv$; P[*w*]/+ and females of the genotype $z^1w^{1118}cv/z^+w^{1118}$; P[*w*]/+ were constructed (*cv* is the genetic abbreviation for the *crossveinless* gene). All have a wild-type phenotype (do not interact with z^1 when present in a single copy) with the exception of the two transposons located in 92B, which had a red-brown eye color in the females. This eye color is indicative of a weak interaction.

Flies that are hemizygous or homozygous for z^1w^- and carry either one or two copies of the A^R4-24 transduced *white* gene have similar phenotypes.

Intercalary heterochromatin may exist within otherwise euchromatic regions. However, 24CD has not been noted as a site of intercalary heterochromatin [E. V. Ananiev *et al.*, *Chromosoma* (Berl.) **70**, 1 (1978)]. Furthermore, there is no evidence for the induction by intercalary heterochromatin of mosaic position effects on rearranged euchromatic genes (*1*). We cannot now rule out the possibility that the P-*white* transposon in A^R4-3 may have carried flanking sequences with it in its transposition to 24CD. We have shown a difference in the distance between the transposon and the next adjacent Bam HI site in flanking DNA on one side of the sites of insertion in A^R4-3 and A^R4-24 (*12*). However, we have not proved that the DNA flanking the insertion in A^R4-24 originates from the 24CD region.

22. Transduced *white* genes may also be more sensitive to position effects in their interaction with z^1 than are rearranged *white* genes that are part of larger translocations (*5*).
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25. We thank T. Laverty for performing the in situ hybridizations. Supported by the Damon Runyon–Walter Winchell Foundation and the American Cancer Society (T.H.) and a grant from the National Institutes of Health.

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Hepatitis B Virus DNA Sequences in Lymphoid Cells from Patients with AIDS and AIDS-Related Complex

Abstract. *A lymphotropic virus HTLV-III/LAV was recently identified as the etiologic agent of the acquired immune deficiency syndrome (AIDS). In a study of concomitant hepatitis B infections in patients with AIDS or the AIDS-related complex, DNA sequences of hepatitis B virus (HBV) were found in fresh and cultured lymphocytes from patients with AIDS even in the absence of conventional HBV serological markers. Furthermore, the restriction DNA pattern was consistent with the integration of the viral DNA. These results should prompt additional studies to reevaluate a possible role of HBV as a cofactor in AIDS in addition to the HTLV-III/LAV causal agent.*

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A human T-lymphotropic retrovirus (HTLV-III, LAV) (*1–5*) has been isolated from patients with the acquired immune deficiency syndrome (AIDS) or AIDS-related complex (ARC) and from clinically asymptomatic individuals (*3, 6*). This virus has been causally related to AIDS and ARC, and it appears to be a necessary etiologic agent of these syndromes (*7*). However, infection by HTLV-III/LAV induces different responses depending on the individuals, suggesting that one or more other factors, such as hepatitis B virus (HBV), cytomegalovirus (CMV), or Epstein-Barr virus (EBV) (*8*), might also have a role in the pathogenic mechanism that leads to immune deficiency or might enhance the likelihood of disease manifestations of HTLV-III/LAV infection. Indeed, on the basis of epidemiological and biological considerations, several authors have proposed an etiologic role for HBV (*9*). Serological markers of HBV have frequently been detected in patients with AIDS and in people at high risk for AIDS (for example, homosexual men, hemophiliacs, and intravenous drug abusers), and HBV DNA sequences have been identified in bone marrow cells (*10*) and mononuclear blood cells of such subjects (*11, 12*). These data prompted us to investigate concomitant hepatitis B infections in patients with AIDS and ARC. We report here that HBV DNA sequences were present in lymphocytes derived from AIDS patients who were serologically HBV-positive or HBV-negative. In the lymphocyte populations assayed, we also detected a common pattern consistent with the integration of the HBV DNA and with free monomeric viral forms.

Lymphoid cell DNA from four patients, two with AIDS (one of them with Kaposi's sarcoma) and two with ARC, were assessed. That each of the patients was infected with HTLV-III/LAV was demonstrated by (i) the presence of antibodies to HTLV-III in the serum samples detected by indirect immunofluorescence as described (*13*), (ii) the identification of reverse transcriptase activity (RT) in the supernatant of primary mononuclear cell cultures and H9 cocultures, and (iii) the detection of HTLV-III antigens p15 and p24 in acetone-fixed cultured lymphocytes by indirect immunofluorescence in the presence of specific monoclonal antibodies (*1, 14*).

Serological markers for HBV were