has also been observed in *S. columbianus* [A. L. Steiner, *J. Mammal.* **53**, 601 (1972)] and *S. parryii* [I. G. McClean, *Anim. Behav.* **31**, 32 (1983)], both of which are probably harem-polygynous.
24. B. C. R. Bertram, *J. Zool.* **177**, 463 (1975).
25. The evicted mother and one evicted juvenile were never seen again and presumably died. The other evicted juvenile incorporated herself into an adjacent coterie where she weaned a litter 2 years later; no other prarie dog at the study colony ever permanently transferred into a different coterie so soon after weaning.
26. L. H. Frame, J. R. Malcolm, G. W. Frame, H. van Lawick, *Z. Tierpsychol.* **50**, 225 (1979); J. P. Rood, *Anim. Behav.* **28**, 143 (1980); see also C. Packer and A. E. Pusey, in (*1*), pp. 31–42.
27. The colony less than 5 years old contained approximately 45 adults and yearlings and food that was more abundant and of higher quality than food at the study colony [M. G. Garrett, J. L. Hoogland, W. L. Franklin, *Am. Midl. Nat.* **108**, 51 (1982)]. The colony less than 15 years old contained more than 500 adults and yearlings and probably also contained more and better food than did the study colony.
28. J. V. Tileston and R. R. Lechleitner, *Am. Midl. Nat.* **75**, 292 (1966).
29. Coterie members restrict feeding and other activities to the home coterie territory which does not usually fluctuate in size (*8, 15*). Thus, overcrowding within coteries could occur even though the colony as a whole is not overcrowded. Because infanticide sometimes occurs in small coteries with large territories, overcrowding within coteries evidently is not the only reason why lactating females sometimes kill the offspring of close kin. Prairie dogs today occupy less than 10 percent of their former geographic range [T. W. Clark, *Natl. Geogr.* **156**, 270 (1979)], but it is not known whether overcrowding is more serious in South Dakota populations now than in populations over evolutionary time.
30. Black-tail females may sometimes kill their own offspring. This infanticide would be in addition to the 51 percent shown in Table 1. It would be difficult to distinguish between infanticide perpetrated by the mother and that by an undetected marauder.
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33. Within coteries, there is no evidence that marauders are more likely to kill offspring of distant kin rather than offspring of close kin. Individual prairie dogs also do not discriminate between close kin and distant kin in behavioral interactions [J. L. Hoogland, *Anim. Behav.* **34**, 263 (1986)] or when giving an alarm call (*12*).
34. R. L. Mumme, W. D. Koenig, F. A. Pitelka, *Nature (London)* **306**, 583 (1983).
35. W. D. Hamilton, *Annu. Rev. Ecol. Syst.* **3**, 193 (1972).
36. I thank the 94 field assistants who have helped me during the last 12 years, and I especially thank T. Anderson, J. Hoogland, M. Hoogland, P. Hardison, P. McDonald, S. Partan, and P. Walsh. I also thank the staff at Wind Cave National Park, especially L. McClanahan, J. Randall, R. Klukas, L. Butts, and S. Pierce. P. Sherman's thorough study of infanticide in Belding's ground squirrels triggered field assistants and me to look so seriously for infanticide in prairie dogs. For discussion and help with the manuscript, I thank R. Alexander, J. Bonner, C. Brown, C. Crockett, B. DeLanoy, P. Grant, P. Harvey, G. Hausfater, H. Horn, S. Hrdy, U. Huck, J. King, W. Koenig, D. Mock, A. Pusey, D. Rubenstein, P. Sherman, W. Shields, D. Wilcove, G. Williams, E. Wilson, and R. Wrangham. Supported by NSF grants BNS77-15594, BNS79-24093, DEB81-02791, and BSR83-07705, National Geographic Society grants 78-1860, 79-2063, and 84-2776, the American Philosophical Society, the Center for Field Research, the Eppley Foundation for Research, the Max McGraw Wildlife Foundation, the Whitehall Foundation, and the universities of Maryland, Michigan, and Minnesota.

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Rescue of the *Drosophila* Phototransduction Mutation *trp* by Germline Transformation

Abstract. *Phototransduction is the process by which light-stimulated photoreceptor cells of the visual system send electrical signals to the nervous system. Many of the steps that follow the initial event in phototransduction, absorption of light by rhodopsin, are ill-defined. The fruitfly,* Drosophila melanogaster, *provides a means to dissect phototransduction genetically. Mutations such as* transient receptor potential (trp) *affect intermediate steps in phototransduction. In order to facilitate molecular studies of phototransduction, the* trp *gene was isolated and its identity was confirmed by complementing the mutant* trp^{CM} *allele of the* trp *gene by P-element mediated germline transformation of a 7.1-kilobase DNA fragment. Expression of the* trp *gene begins late in pupal development and appears to be limited to the eyes and ocelli.*

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Phototransduction is a neuronal excitation process that is stimulated by light and results in a change in the flow of ions across the photoreceptor membrane. Physiological studies suggest that several important features of the phototransduction process are similar in vertebrates and invertebrates. In both systems, the pigments that absorb light and undergo photoisomerization are protein-chromophore complexes known as rhodopsins (*1*). It appears that, in vertebrates and invertebrates, there are a series of intermediate steps between the photoisomerization of the visual pigments and the change in permeability of the photoreceptor membrane (*2*). With the exception of vertebrate transducin (*3*), little is known in either system about the proteins that function in the intermediate phototransduction events.

The fruitfly *Drosophila melanogaster* is an excellent system in which to study phototransduction. The anatomical features of the fruitfly's compound eyes have been described in detail, mutations in genes affecting phototransduction have been isolated (*2*), and techniques for germline transformation have been developed (*4*). Many of the *Drosophila* mutants believed to intervene in phototransduction were isolated on the basis of abnormal electroretinogram (ERG) recordings (*2*). ERG's measure the change in potential across the photoreceptor membrane in response to light. Wild-type flies display a sustained receptor potential that decays very gradually during continuous bright illumination. Within 5 seconds of cessation of a light stimulus (dark recovery) the response of wild-type flies to a subsequent stimulus is nearly maximal. Of the mutants thought to affect an intermediate step in phototransduction, the transient receptor potential (*trp*) mutant is among the most intensively studied (*5–8*). The *trp* mutant is characterized by a rapid decay of the receptor potential during illumination with bright light (*5*). It also displays an abnormally slow dark recovery (*5*). Despite many detailed studies, the basis of the *trp* phenotype is not well understood. The *trp* phenotype is not due to quantitative or qualitative changes in the photopigment (*6*). Minke has proposed that the rapid decay of the receptor potential is caused by a light-induced reduction in excitation efficiency resulting from an unknown defect in an intermediate stage of phototransduction (*6*).

Currently, none of the genes encoding products relevant to an intermediate step in phototransduction have been isolated in *Drosophila*. A more thorough understanding of the processes disrupted by the *trp* mutation and other phototransduction mutations would be facilitated by identification and characterization of the corresponding genes and gene products. We now describe the identification of *trp* by a germline transformation procedure that rescues the phenotype (*9*). The developmental expression and tissue localization of the *trp* RNA are also presented.

A gene encoding a product that functions specifically in an intermediate process in phototransduction might be expected to be expressed exclusively in the eyes. Therefore, the criteria used to identify a gene likely to rescue the *trp* phenotype were that it map to the cytogenic position of the *trp* mutation and that it be expressed specifically in the eye. A differential screen of a library of cloned *Drosophila* genomic DNA segments with polyadenylated [poly(A)$^+$] RNA prepared from fly heads and bodies yielded 20 cloned sequences that were expressed more abundantly (by a factor of 10 at least) in the head than in the body (*10*). Among these 20, one (λ559)

mapped cytogenetically by in situ hybridization to the same position as the *trp* mutation, 99C, which is near the tip of the third chromosome (*10, 11*).

The spatial distribution of transcripts homologous to λ559 was determined by in situ hybridization to tissue sections. The DNA fragment used to probe for the 559 RNA was identified by hybridizing restriction endonuclease–digested λ559 DNA on nitrocellulose filters to a ^{32}P-labeled complementary DNA (cDNA) probe that was prepared from polyadenylated RNA of fly heads and bodies. No λ559 fragments hybridized to body cDNA and a single 1.7-kilobase (kb) Eco RI fragment hybridized to head cDNA. This 1.7-kb Eco RI fragment was subcloned from λ559 to pUC13 (*12*) to create the plasmid p559E1.7, which was labeled with tritiated nucleotides by nick translation and hybridized to tissue sections of adult fly heads. The tissue distribution of the head-specific 559 RNA is shown in Fig. 1. The hybridization of the DNA probe occurred predominantly in the eye, consistent with the localization expected for *trp* RNA. Few grains were localized in the pigment cell nuclei just below the cornea, suggesting that 559 is not expressed in the pigment cells and may be localized exclusively to the retinula cells. The retinula cells are the specialized cells in which phototransduction occurs. In addition to the compound eyes, *Drosophila* has three small visual organs, ocelli, located at the vertex of the head. The ocelli are not included in the section in Fig. 1. However, examination of the other sections that include portions of the ocelli showed that *trp* is also expressed in these small visual organs. This result is consistent with the previous finding that the *trp* mutation also disrupts the functioning of the ocelli (*13*).

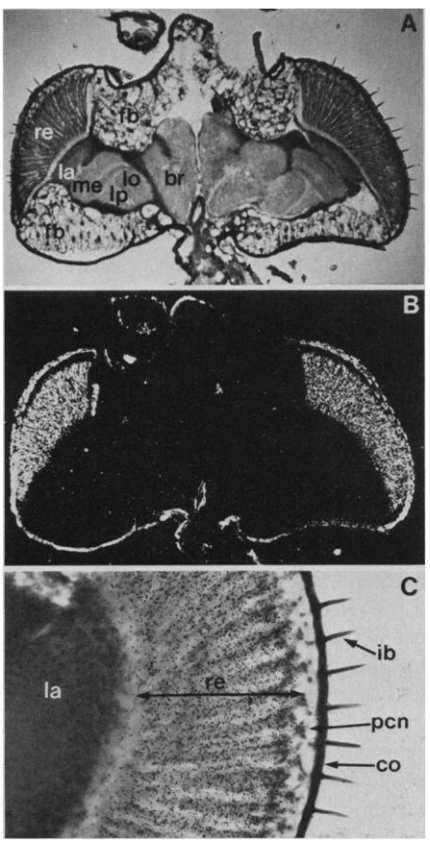

Fig. 1. Spatial distribution of the putative *trp* RNA in adult fly heads. Horizontal sections (8 μm) of adult heads (Canton S) were prepared and hybridized to p559E1.7 that had been labeled with ^{3}H as described (*20*). (A) Bright field. Fly head is approximately 0.6 × 0.35 mm. (B) Dark field. Same section as (A). (C) Bright field. Higher magnification of compound eye. Br, brain; co, cornea; fb, fat bodies; ib, interommatidial bristles; la, lamina ganglionaris; lo, lobula; lp, lobula plate; me, medulla; pcn, pigment cell nuclei; re, retinular cells. Localization of grains to the surface of the eye was probably a result of nonspecific binding to cuticular structures often observed with DNA probes (*20*). In the dark-field image (B), some light scattering by the cuticle is also seen.

Expression of the 559 RNA during *Drosophila* development was also investigated. If the 559 transcripts correspond to the *trp* gene, then expression might be limited to the last stages of *Drosophila* development and to the adult (when phototransduction occurs). When p559E1.7 was used as the probe, a single 4.2-kb RNA was detected in adult RNA (Fig. 2, lane A). This 4.2-kb RNA was also observed in the lane containing adult head RNA but not in the lane containing adult body RNA, even though only one-eighth the amount of adult head RNA was loaded onto the gel. We will refer to this 4.2-kb RNA as the putative *trp* messenger RNA. A sixfold longer exposure of this autoradiogram did not reveal any RNA homologous to the probe in the adult body. This observation is consistent with the results of dot blot experiments by Levy *et al.*, who reported that 559 RNA is at least 26 times more abundant in the head than in the body (*10*). Thus, within the resolution of the RNA blot and in situ hybridization, we conclude that expression of the putative *trp* mRNA is limited to the eyes and ocelli. We estimate that if this RNA were expressed in the body at 0.1 percent of the level observed in the head then a band would have been detected in the longer exposure of this autoradiogram. Expression of the putative *trp* RNA is limited to the last stages of development. The putative *trp* RNA was detected only in the adult head and in pupae collected less than 24 hours prior to eclosion. Thus, the cytogenetic position, tissue specificity, and timing of expression of the 4.2-kb RNA are consistent with the 559 DNA encoding some portion of the *trp* gene.

To determine whether the DNA encoding the 4.2-kb RNA corresponds to the *trp* gene we identified the coding region and used a DNA fragment encompassing the transcribed region to rescue the mutant phenotype by germline transformation. Since the λ559 clone encodes a maximum of 1.7 kb of coding region for the 4.2-kb putative *trp* RNA, we isolated an overlapping bacteriophage clone, λ559B, from a *Drosophila* genomic library (*14*). The λ559B DNA was digested with restriction endonucleases, fractionated on agarose gels by electrophoresis, blotted to duplicate nitrocellulose filters,

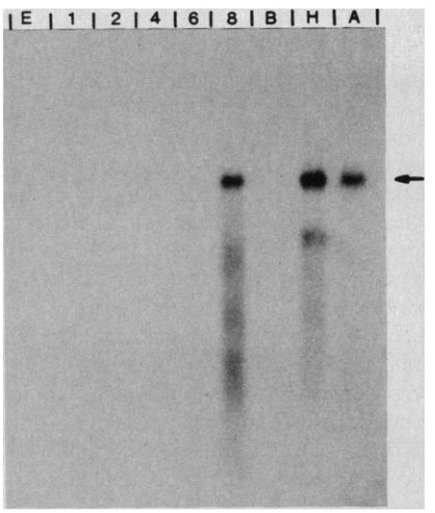

Fig. 2. Expression of the putative *trp* RNA during *Drosophila* development. Oregon R,P2–strain embryos were collected for 4 hours and allowed to develop at 25°C for 0 to 16 hours (embryos) or for 1, 2, 4, 6, 8, and 9 to 10 days (adults). Heads and bodies of adults were separated by agitation of frozen flies (*21*). The 1-, 2- and 4-day collections coincided approximately with the first, second, and third larval instar stages of development. The 6- and 8-day collections coincided approximately with the early and late pupal stages. RNA was prepared (*22*) and polyadenylated RNA was fractionated by affinity chromatography with oligo(dT) cellulose (*23*). Adult head poly(A)$^+$ RNA (0.25 μg) and poly(A)$^+$ RNA (2.0 μg) for all other samples were fractionated on a 3 percent formaldehyde + 1.5 percent agarose gel (*24*), blotted to nitrocellulose, probed with p559E1.7 that had been nick-translated with ^{32}P, and visualized by autoradiography. Lanes are designated as follows: E (0- to 20-hour embryos); 1, 2, 4, 6, 8 (days of development); B (adult bodies); H (adult heads); and A (whole adults). The arrow indicates the position of the major 4.2-kb band. Lambda DNA digested with Hind III and 3′ end-labeled with ^{32}P was used as the size marker (not shown). The absence of RNA bands in the other lanes was not due to RNA degradation or poor transfer to the nitrocellulose since the expected bands were detected upon probing the filter again with a *Drosophila* actin DNA probe, pDmA2 (*25*).

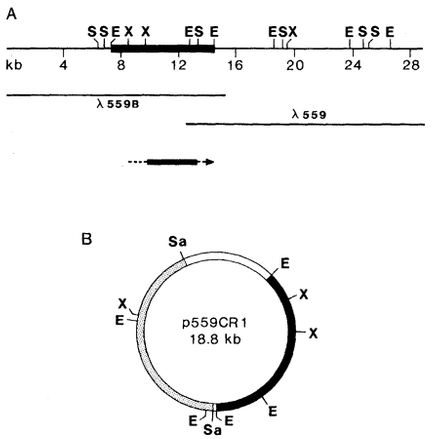

Fig. 3. Physical maps of the putative *trp* genomic region and transformation vector. (A) Physical maps of genomic DNA contained within λ559 and λ559B. Top horizontal line represents the *Drosophila* genomic DNA encompassing the sequences encoding the putative *trp* mRNA. The bold portion of this line indicates the 7.1-kb genomic DNA segment that was cloned into the P-element transformation vector (Fig. 3B). The Sac I (S), Eco RI (E), and Xho I (X) cleavage sites are shown. The genomic DNA in the λ clones are represented by the horizontal lines labeled λ559 and λ559B. The bottom line indicates the coding region for the putative *trp* mRNA. The arrowhead indicates the 3′ end of the RNA. The broken lines adjacent to both ends of the bold solid line indicate the limits of the region within which we have mapped the 5′ and 3′ ends of the RNA. (B) Map of transformation vector. The unshaded segment represents the 3.6-kb Carnegie 3 (*16*) portion of p559CR1. The 7.1-kb Eco RI fragment (7.3 to 14.4 kb) subcloned from λ559B is indicated by the fully shaded region. The stippled region represents the 8.1-kb Sal I ry^+ fragment (*17*). The Sal I (Sa), Eco RI (E), and Xho I (X) sites are indicated.

and probed with ^{32}P-labeled cDNA prepared from head and body RNA of adult flies. This analysis indicated that the putative *trp* RNA is encoded within a 6.0-kb DNA segment extending from the Xho I site at 8.4 kb to the Eco RI site at 14.4 kb (Fig. 3A). A second RNA, expressed exclusively in bodies, is transcribed from the region to the left of the Eco RI site at 7.3 kb. The direction of transcription of the putative *trp* RNA was determined by fractionating adult-head RNA on a formaldehyde gel, transferring it to nitrocellulose, and hybridizing it to strand-specific ^{32}P-labeled RNA probes (*15*).

To rescue the *trp* phenotype by germline transformation (*4*), the head-specific 559 gene was subcloned into a P-element transformation vector (*16*). A 7.1-kb fragment generated by partial cleavage of λ559B with Eco RI (7.3 to 14.4 kb) was subcloned into the P-element–mediated gene transfer vector Carnegie 3 (*16*) to create the plasmid p559C3A. To provide a visible marker for easy scoring of transformants, an 8.1-kb Sal I fragment containing the $rosy^+$ (ry^+) gene (*17*) was cloned into p559C3A to create p559CR1 (Fig. 3B). Rescue of the mutant ry^{506} phenotype would result in restoration of a wild-type eye color. Transformants exhibiting a wild-type eye color would also contain the 559 DNA and could be tested for rescue of the *trp* phenotype.

Six independent ry^+ transformants with inserts mapping to the X or second chromosome were crossed into a trp^{CM} background (*18*) and tested for rescue of the *trp* phenotype on the basis of electroretinogram recordings (ERG). The ERG's were performed by placing a recording electrode on the cornea and a reference electrode in either the abdomen, thorax, or proboscis and then illuminating the eye with white light. The light-induced electrical response due to current flow in the extracellular medium of the eye was recorded on an oscilloscope. A typical ERG recording induced with a white-light stimulus from ry^{506} flies is shown in Fig. 4A. This ERG is indistinguishable from that of wild-type flies. The transient spikes of the ERG

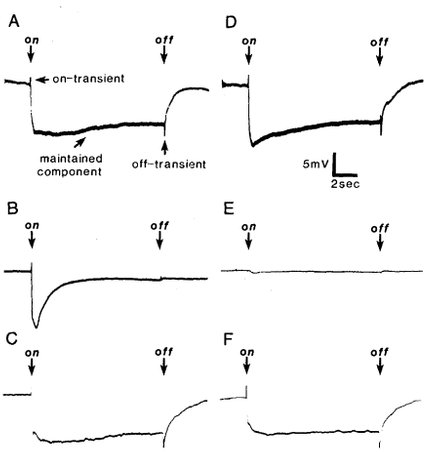

Fig. 4. Electroretinogram recordings of ry^{506} (A and D), $ry^{506}trp^{CM}$ (B and E) and a $ry^{506}trp^{CM}$ fly transformed with p559CR1 (P[*ry*,559]5) (C and F). Responses were to a white-light stimulus of 12-second duration after flies were in the dark for 2 minutes (A to C) or 5 seconds (D to F) since cessation of the previous light stimulus. The ERG recordings of ry^{506} and $ry^{506}trp^{CM}$ flies were indistinguishable from Canton S wild-type and trp^{CM} flies, respectively (not shown). Five other transformed lines, P[*ry*,559]1, P[*ry*,559]3, P[*ry*559]7, P[*ry*,559]9, and P[*ry*,559]11 gave responses indistinguishable from P[*ry*,559]5. The initiation (on) and cessation (off) of the light stimulus are indicated. The maintained component and the on- and off-transients are identified in (A). Signal amplitude and time scale are as indicated by the scale bars.

coincident with turning the light stimulus on and off are thought to originate postsynaptically to the photoreceptors in the second-order cells of the lamina ganglionaris (*2*). The maintained component is believed to be the summation of the membrane potentials of the photoreceptor cells. In wild-type or in ry^{506} flies, the maintained component decayed very gradually before returning to the baseline (resting potential). This feature of the maintained component was different in trp^{CM} flies. Both trp^{CM} and ry^{506} trp^{CM} flies displayed a normal on-transient, but the maintained component quickly decayed close to the baseline (Fig. 4B). The dark recovery of trp^{CM} flies was also abnormal. The response of wild-type flies to light stimulus was nearly maximal 5 seconds after cessation of the preceding light stimulus (Fig. 4D). However, there was little recovery in trp^{CM} flies after 5 seconds (Fig. 4E). The mutant recovered gradually, achieving about 90 percent of the maximum response after 60 seconds in the dark (*5, 6*). Of primary importance, both a wild-type decay rate of the maintained component and wild-type dark recovery were restored in trp^{CM} flies that had been transformed with p559CR1 (Fig. 4, C and F). All six independent ry^+ transformants tested displayed a trp^+ phenotype. Therefore, the *trp* gene is contained in the 7.1-kb 559 fragment that had been cloned into p559CR1 (*19*).

Despite numerous physiological studies over the last 16 years, the role of the *trp* product in phototransduction has been elusive. The identification of the *trp* gene will facilitate a molecular analysis of the *trp* function in phototransduction. The sequences required to code for the 4.2-kb RNA and for function of the *trp* gene were localized to a 7.1-kb fragment that rescues the *trp* phenotype. Moreover, we have shown that the *trp* product is specific to the visual organs and is expressed beginning late in development shortly before phototransduction begins in the adult visual system.

We have presented a useful approach for identifying DNA sequences likely to encode phototransduction genes. This strategy entails screening for DNA sequences that are transcribed preferentially in the eye and that map to the cytogenetic position of a phototransduction mutant. Confirmation that the DNA sequences does encode the corresponding phototransduction gene can then be obtained by testing for complementation of the mutant phenotype after introduction of the cloned DNA into the genome by P-element–mediated gene transfer.

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18. The 924 ry^{506} embryos with the M cytotype were injected (*4*) with p559CR1 (400 mg/ml) and pπ25.7wc (50 mg/ml) and gave rise to 144 fertile adults (G0). The 144 G0 adults were mated to homozygous ry^{506} flies and the G1 flies were scored for ry^+ eye color. One or more progeny of 14 G0 individuals exhibited wild-type eye color and were, therefore, successful transformants. The strain ry^{506} is homozygous for a deletion of a segment of the XDH-coding region and therefore cannot revert to ry^+ (provided by W. Bender, unpublished results). Balanced stocks of ry^+ P-element insertions were established. Six transformants with inserts mapping to the X (two transformants) or second chromosomes (four transformants) were crossed to a strain containing the third chromosome balancer TM3, p^p e^s ry^{RK} bx^{34e} *Sb* trp^+ (isolated by R. Karess, unpublished). The P[*ry*,559]; TM3, ry^{RK}*Sb* trp^+/ry^{506} trp^+ flies were crossed to $ry^{506}trp^{CM}$ flies and their P[*ry*, 559]; TM3, ry^{RK}*Sb* $trp^+/ry^{506}trp^{CM}$ progeny were backcrossed again to the $ry^{506}trp^{CM}$ strain. Two ry^+Sb^+ flies (P[*ry*,559]; $ry^{506}trp^{CM}/ry^{506}trp^{CM}$) derived from each of the six transformants were tested for rescue of the *trp* phenotype. Two $ry^{506}Sb^+$ siblings ($ry^{506}trp^{CM}/ry^{506}trp^{CM}$) of each of the six transformants were also tested. These latter flies served as negative controls. All flies containing the P[*ry*,559] insertion were trp^+ in phenotype and all $ry^{506}Sb^+$ siblings tested that lacked the P-element insertion gave ERG recordings indistinguishable from the $ry^{506}trp^{CM}$ flies. The eight transformants with third chromosome inserts were not tested for rescue of the *trp* phenotype since the *trp* gene is also on the third chromosome. Consequently, these transformants could not be moved into a trp^{CM} background as readily as the transformants with X and second chromosome inserts.
19. Starting from the λ559 clone, Wong *et al.* isolated a series of overlapping λ clones that span the breakpoints in 99C5-6 to which *trp* has been mapped (*11*). These workers probed RNA blots with bacteriophage DNA that had been labeled with ^{32}P, and reported that the stretch of DNA represented by these clones encodes four RNA species in Oregon R, two of which were missing in trp^{CM}. We have prepared RNA from the same fly stocks (Oregon R and trp^{CM}), fractionated the poly(A)$^+$ RNA on a formaldehyde gel, blotted to nitrocellulose, and probed with *trp* genomic DNA (p559E1.7) that had been labeled with ^{32}P. The results of this analysis showed that the size and concentration of *trp* RNA were similar in both fly stocks.
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22. Total (nuclear and cytoplasmic) RNA was prepared by homogenizing 3 grams of tissue in 10 ml buffer (0.15*M* sodium acetate, 5 m*M* EDTA, 1 percent sodium dodecyl sulfate, 50 m*M* tris-HCl, *p*H 9.0), 5 ml of phenol, and 5 ml of a mixture of chloroform + isoamyl alcohol (100:1) by means of a Brinkmann Polytron. The homogenate was extracted once with chloroform + isoamyl alcohol, four times with phenol + chloroform (1:1), and once with chloroform + isoamyl alcohol, and was then precipitated in ethanol.
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Amino Acid Homology Between the Encephalitogenic Site of Myelin Basic Protein and Virus: Mechanism for Autoimmunity

Abstract. *Amino acid sequence homology was found between viral and host encephalitogenic protein. Immune responses were then generated in rabbits by using the viral peptide that cross-reacts with the self protein. Mononuclear cell infiltration was observed in the central nervous systems of animals immunized with the viral peptide. Myelin basic protein (MBP) is a host protein whose encephalitogenic site of ten amino acids induces experimental allergic encephalomyelitis. By computer analysis, hepatitis B virus polymerase (HBVP) was found to share six consecutive amino acids with the encephalitogenic site of rabbit MBP. Rabbits given injections of a selected eight– or ten–amino acid peptide from HBVP made antibody that reacted with the predetermined sequences of HBVP and also with native MBP. Peripheral blood mononuclear cells from the immunized rabbits proliferated when incubated with either MBP or HBVP. Central nervous system tissue taken from these rabbits had a histologic picture reminiscent of experimental allergic encephalomyelitis. Thus, viral infection may trigger the production of antibodies and mononuclear cells that cross-react with self proteins by a mechanism termed molecular mimicry. Tissue injury from the resultant autoallergic event can take place in the absence of the infectious virus that initiated the immune response.*

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Many viruses share antigenic sites with normal host cell components (*1–3*), a phenomenon known as molecular mimicry. This commonality has been demonstrated by direct comparisons of amino acid sequences. Moreover, the finding of monoclonal antibodies that react with both host and virus constituents suggests that viruses have the potential to trigger autoimmune responses and resultant disease. Antibodies or cytotoxic lymphocytes generated against the virus might cross-react with self proteins, thus causing cellular injury leading to disease. Once the infectious agent initiates this process, it need not be present during the autoimmune destruction that follows.

Molecular mimicry occurs frequently and with various DNA and RNA viruses. Lane and Hoeffler (*3*), using a monoclonal antibody, showed that the large T antigen of simian virus 40 and normal proteins of host cells have common antigenic sites. Subsequently, we found that the measles virus phosphoprotein (P3), the 140-kilodalton (kD) protein of herpes simplex virus, and the hemagglutinin of vaccinia virus all reacted with distinct epitopes on intermediate filaments (*1*). In a study of more than 600 monoclonal antibodies to 11 different viruses, Srini-