The Drosophila rhodopsin cytoplasmic tail domain is required for maintenance of rhabdomere structure

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ABSTRACT The ninaE-encoded Rh1 rhodopsin is the major light-sensitive pigment expressed in Drosophila R1–6 photoreceptor cells. Rh1 rhodopsin localizes to and is essential for the development and maintenance of the rhabdomere, the specialized membrane-rich organelle that serves as the site of phototransduction. We showed previously that the vertebrate bovine rhodopsin (Rho) is expressed and properly localized in Drosophila photoreceptor cells. Drosophila photoreceptors expressing only Rho have normal rhabdomere structure at young ages, but the rhabdomeres are not maintained and show extensive disorganization by 7–10 days of age. A series of Rho-Rh1 opsin chimeric rhodopsins were used to identify Rh1 domains required for maintenance of rhabdomeric structure. The results show that the Rh1 rhodopsin cytoplasmic tail domain, positioned to interact with cytoplasmic structural components, plays a major role in promoting rhabdomeric organization.—Ahmad, S. T., Natochin, M., Artemyev, N. O., O’Tousa J. E. The Drosophila rhodopsin cytoplasmic tail domain is required for maintenance of rhabdomere structure. FASEB J. 21, 449–455 (2007)

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The DROSOPHILA compound eye is composed of ~800 ommatidia arranged in a honeycomb-like pattern. Each ommatidium contains R1–6 photoreceptor cells along with R7 and R8 photoreceptor cells and accessory cells. The R1–6 photoreceptor cells form a slender cylinder that spans the length of the retina. Each photoreceptor cell contains a rhabdomere, a highly organized microvillar structure, projecting toward the center of the cylinder. Rhodopsin, the visual pigment responsible for the initiation of the phototransduction cascade, is the major protein of the rhabdomere. Other proteins present in the rhabdomere include the components of the phototransduction cascade, F-actin (1), the unconventional myosin NINAC (2), and chaoptin, a membrane protein involved in adhesion between the neighboring microvilli (3).

The Rh1 rhodopsin encoded by the ninaE gene is expressed in R1–6 photoreceptor cells (4, 5). One class of ninaE mutations are genetically dominant and show cellular degeneration and cell death. Faulty transport of rhodopsin to the rhabdomeres appears to be the initial defect in most of these mutants (6, 7), though other mechanisms have been implicated in several mutants (8). Other ninaE mutations, genetically recessive, show very low levels of rhodopsin and cause atrophy of the rhabdomeric microvillar structure. The progression of this atrophy depends on the severity of the ninaE mutation, and ranges from poorly developed rhabdomeres at eclosion to progressive loss of rhabdomeric structure over a course of several weeks (1, 9, 10). Thus, rhodopsin not only plays a central role in phototransduction but also is essential for the development and maintenance of the rhabdomeres and photoreceptor cells.

The Drosophila (Rh1) and bovine (Rho) opsins are related with 22% identity in amino acid sequence and 49% conservation of amino acids with similar chemical properties (4, 5). The structural organization in Rho and Rh1 is also conserved. Both possess seven transmembrane helices. There are three intracellular loops and a carboxyl terminal tail projecting into the cytoplasm. The three extracellular loops and an amino terminal tail projects into the extracellular space of Drosophila rhodopsins and within the lumen of the discs of vertebrate rod outer segments. The retinal chromophore binds via a Schiff’s base linkage to a lysine residue in the seventh transmembrane region, with seven of the eleven amino acid residues surrounding the lysine are identical in Rho and Rh1. Other domains of high identity exist in the second extracellular loop and the first and second cytoplasmic loops.

There are some notable differences in Rh1 and Rho structure. The major structural difference is the addition of 12 amino acid residues in the third cytoplasmic loop of Rh1 (4). Rh1 also lacks a counterion (Glu-113 in helix 3 serves as counterion in Rho) to neutralize the protonated Schiff’s base lysine-chromophore linkage. The visual pigment responsible for the initiation of the phototransduction cascade, is the major protein of the rhabdomere. Other proteins present in the rhabdomere include the components of the phototransduction cascade, F-actin (1), the unconventional myosin NINAC (2), and chaoptin, a membrane protein involved in adhesion between the neighboring microvilli (3).

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(11). The retinal chromophore also differs, with Rho binding retinal and Rh1 binding 3-hydroxyretinal (12). The mature form of Rho maintains N-linked glycosylation at Asn-2 and Asn-15 residues in extracellular amino-terminal tail (13), whereas mature Rh1 is deglycosylated (14).

We recently reported expression of bovine Rho opsin in *Drosophila* photoreceptors (15). We show here that this vertebrate opsin is not capable of providing long-term stability to the rhabdomeric organization. By expression of a series of chimeric Rh1-Rho fusion proteins, we demonstrate that the carboxyl-terminal tail of Rh1 contains the major cytoplasmic determinant responsible for Rh1’s role in rhabdomeric development and structure.

**MATERIALS AND METHODS**

**Construction of Rh1-Rho chimeric genes and transgenic flies**

To generate Rh1-Rho chimeras containing single Rho loops C2 and C3 within the *ninaE* gene, polymerase chain reactions (PCR) were carried out on the pMT4-*ninaE* template flanked with EcoRI and Noti sites. A phosphorylated forward primer encoding the amino-terminal half of the corresponding loop, and a reverse primer containing a Noti site was used to amplify the carboxyl-terminal part of the chimera. A forward primer containing an EcoRI site paired with a phosphorylated reverse primer encoding the amino-terminal half of the corresponding loop yielded the amino-terminal part of the chimera. PCR products were ligated followed by digestion of appropriate ligation adducts (~1 kb) with NotI and EcoRI and ligation into the pMT4 vector. All constructs were entirely sequenced to verify proper chimera construction. These Rh1-Rho chimeras were directionally cloned using NotI and C2 and C3 restriction enzyme sites for *ninaE* promoter, *EcoRI* and *NotI* sites paired with a phosphorylated *EcoRI* site paired with a phosphorylated *NotI* site was used to amplify the carboxyl-terminal part of the chimera. PCR products were ligated followed by digestion of appropriate ligation adducts (~1 kb) with *NotI* and *EcoRI* and ligation into the pMT4 vector. All constructs were entirely sequenced to verify proper chimera construction. These Rh1-Rho chimeras were directionally cloned using *EcoRI* and *NotI* restriction sites into a modified pcASpeR4 expression vector (16). In the modified pCaSpeR4 vector, the polylinker region was replaced with a 3.0-kb fragment containing the *ninaE* promoter, *EcoRI* and *NotI* restriction enzyme sites for the cloning of the Rho cDNA, and a 0.7 kb *PstI-HindIII* region corresponding to the 3' untranslated region of *ninaE* gene. Correct chimera construction was confirmed by DNA sequencing, and the element was introduced into embryos of *ninaE* mutant by P-element transformation techniques (17). All flies were reared on standard cornmeal-molasses medium at 25°C under 12-h light-dark conditions unless noted.

**Western blot analysis**

Chimera expression in *Drosophila* was detected by homogenizing 2 or 3 fly heads in Laemmli SDS-sample buffer. Typically, one fly head equivalent was loaded on a 10% SDS-polyacrylamide gel. The proteins were separated using standard electrophoresis techniques and immobilized onto a nitrocellulose membrane. Rho and Rh1 proteins were detected using 1:5000 dilution of 1D4 monoclonal antibody (mAb) and 1:3000 dilution of anti-Rh1 polyclonal antibody (pAb), respectively, in 1× PBS (pH 7.5) containing 0.1% Tween 20 and further processed according to standard protein blotting procedures.

**Immunofluorescence**

For immunological localization, fly heads were embedded in London Resin White (LRW) embedding medium using the following protocol. Fly heads were bisected and fixed in cold 2% glutaraldehyde, 2% formaldehyde solution in 1× PBS (pH 7.5) for 45 min followed by rinsing 3 times for 5 min each in 1× PBS (pH 7.5). Heads were then dehydrated in an ethanol series (50%, 70%, 80% for 10 min each followed by 90% for 30 min). Heads were incubated in 1:1 90% Ethanol: LRW for 15 min then in 100% LRW for 15 min. Heads were transferred to gelatin capsules filled with 100% LRW and sealed tight for polymerization at 50°C overnight. LRW-embedded heads were sectioned (approx. 1 μm thick) and placed on chrome-gelatin subbed slides (1% gelatin, 0.1% chromium potassium sulfate). Head sections were blocked in 2% BSA in 1× PBS (pH 7.5) followed by rinsing 3 times for 5 min each in 1× PBS (pH 7.5). Head sections were incubated with 1:100 dilution of primary antibody (Ab) (1D4 for Rho and C45 mAb for Rh1) for 1 h at room temperature followed by rinsing 3 times for 5 min each in 1× PBS (pH 7.5). Head sections were incubated with 1:100 dilution of anti-mouse Alexa Fluor 488 Ab (Molecular Probes) for 20 min at room temperature followed by rinsing 3 times for 5 min each in 1× PBS (pH 7.5). Stained head sections were mounted in Vectashield antifade mounting medium (Vector Laboratories) and viewed under Nikon Microphot fluorescence microscope attached with a Spot imaging instrument (Diagnostic Instruments).

**RESULTS**

**Rho-expressing flies have defects in rhabdomeric organization**

The *Drosophila* Rh1 rhodopsin is essential for development and maintenance of the rhabdomeric membranes of photoreceptor cells. Figure 1A contains a cross section of *Drosophila* ommatidium showing the large rhabdomeres of the R1–6 cells in a three-day-old wild-type (WT) fly. The rhabdomere size and structure are compromised in the Rh1 null mutant *ninaE* at the same age (Fig. 1B). Expression of the bovine Rho rhodopsin in the Rh1 null background rescued the
small rhabdomeres phenotype observed due to the absence of Rh1 rhodopsin at 3 days posteclosion (Fig. 1C). However the rhabdomeres were smaller and slightly irregular in shape compared to WT flies. More substantial defects in rhabdomeric packing were observed in the Rho flies at 7 days posteclosion (Fig. 1D), and the severity of this phenotype further intensified at 14 days (Fig. 1E). At 14 days, the rhabdomeric membranes were extensively unraveled and occupied a greater area of the photoreceptor cell body. No defects in rhabdomere packing were observed in older flies expressing the Rh1 rhodopsin (data not shown). Unlike Rh1, Rho dissociates into the apoprotein opsin and retinal following light stimulation. We analyzed the rhabdomeric ultrastructure of Rho-expressing flies reared in constant darkness to determine whether this process contributes to the severe unraveling observed in Rho flies. Figure 1F shows the rhabdomeric membranes of these dark-reared photoreceptors also show the severe unraveling phenotype. Thus, the rhabdomeric unraveling phenotype is not due to the conformational changes in Rho brought about by light-triggered dissociation of chromophore. Also, Rho remained localized on the disorganized rhabdomeric membranes found in the photoreceptor cell body of older flies (Fig. 1G), indicating that the lack of rhabdomeric integrity is not due to the absence of Rho protein.

Rh1 cytoplasmic loops C2 and C3 are not required for the maintenance of rhabdomeric integrity

To determine the specific role of Rh1 domains in the maintenance of rhabdomeric integrity we expressed a series of chimera proteins combining the cytoplasmic loops of Rh1 and Rho opsin in various combinations in transgenic flies. These proteins were expressed exclusively in the R1–6 cells by driving their expression with the Rh1 promoter and were the only rhodopsin expressed in these photoreceptors because all transgenes were placed in the Rh1 null ninaE17 background.

Transgenic flies expressing the RhoC2-Rh1 and RhoC3-Rh1 chimeric proteins contained the substitution of the second (Fig. 2A, D) and third (Fig. 2E, H) Rh1 cytoplasmic loops, respectively, with the corresponding Rho loops. These chimeric protein levels were comparable to the WT Rh1 levels (Fig. 2B for RhoC2-Rh1 and Fig. 2F for RhoC3-Rh1). Immunolocalization analysis showed that RhoC2-Rh1 (Fig. 2C) and RhoC3-Rh1 (Fig. 2G) chimera proteins are localized to R1–6 rhabdomeres, identical to the localization of Rh1 (data not shown). Absence of staining in R7 cell (arrows in Fig. 2C and G) served as a control for antibody specificity and exclusive expression of the transgene within R1–6 photoreceptors.

The ultrastructural morphology of rhabdomeres in flies expressing RhoC2-Rh1 and RhoC3-Rh1 chimeras is presented in Fig. 3. At 7 days posteclosion, flies expressing RhoC2-Rh1 (Fig. 3A) and RhoC3-Rh1 (Fig. 3B) showed excellent rhabdomeric morphology, comparable to the WT flies shown in Fig. 1A. The ommatidial structure remained intact at 14 days posteclosion in both RhoC2-Rh1 (Fig. 3D) and RhoC3-Rh1 (Fig. 3E)

Figure 1. Rhabdomere disintegration in Rho flies. Electron micrograph of a cross section through the distal portion of the retina from WT at three days (A) shows the outer R1–6 and the centrally located R7 rhabdomeres, ninaE17 (Rh1 null) at three days (B) shows severely reduced R1–6 rhabdomeres. R7 rhabdomere is unaffected because it does not express Rh1 rhodopsin and expresses Rh3 or Rh5 rhodopsin. (C–F) Similar electron micrographs of transgenic fly expressing Rho in Rh1 null genetic background at three days (C), 7 days (D), 14 days (E), and 14 days when reared in dark (F). G) Confocal image of a LRW-embedded section of retina of the Rho-expressing fly at 14 days probed with anti-Rho Ab. Scale bar = 5 μm.

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chimera-expressing flies. The lack of any structural abnormality in rhabdomeres indicates that Rh1 cytoplasmic loops C2 and C3 do not play a major role in the maintenance of rhabdomeric integrity.

Rh1 cytoplasmic tail (Ct) is essential for the maintenance of rhabdomeric size

Transgenic flies with the RhoCt-Rh1 chimera, possessing the cytoplasmic tail of Rho (Fig. 2I, L), showed strong rhodopsin expression (Fig. 2J). The RhoCt-Rh1 protein properly localized to R1–6 rhabdomeres (Fig. 2K). At 7 days posteclosion, the rhabdomeres of flies expressing RhoCt-Rh1 chimera (Fig. 3C) were reduced in size and irregular in shape as compared to the WT, RhoC2-Rh1, and RhoC3-Rh1 rhabdomeres. These differences were also evident at 14 days posteclosion, with some photoreceptors lacking a visible rhabdomere in a given section (Fig. 3F). These findings suggested a role of the Rh1 cytoplasmic tail in establishing and maintaining rhabdomeric shape and volume.

We reasoned if the lack of the Rh1 cytoplasmic tail in RhoCt-Rh1 chimera is responsible for the observed defects in rhabdomeric structure, then expression of the reciprocal rhodopsin chimera, i.e., Rh1Ct-Rho containing only the Rh1 cytoplasmic tail (Fig. 4A), should rescue the phenotype. Immunoblot analysis of the head homogenates of flies expressing the Rh1Ct-Rho transgene showed high expression levels of this chimeric protein (Fig. 4B). The chimeric protein properly localized to the rhabdomeres (Fig. 4C). Figure 4D–G compares the structure of Rho and Rh1Ct-Rho photoreceptors at 7 days and 14 days. As predicted, the R1–6 rhabdomeric structures are much improved in the Rh1Ct-Rho photoreceptors at both time points. Thus, the carboxyl-terminal domain of Rh1 on the Rho rhodopsin provides a major determinant in allowing the regular packing of the rhabdomeric membranes.

DISCUSSION

Transgenic Drosophila expressing bovine rhodopsin show high expression levels, proper trafficking and localization in Drosophila photoreceptor cells (15). We show here that Rho is unable to maintain rhabdomeric integrity during the adult life span (Fig. 1). Degeneration is limited to the rhabdomeres and does not lead to photoreceptor cell death. The continued presence of Rho protein on the unraveling membranes shows that the atrophy of the rhabdomeric membranes is not due to lack of Rho on these membranes. Also, the proper localization of Rho to rhabdomeres with no apparent accumulation in the cell body in young flies indicates that the accumulation of Rho-laden membranes in the cell body of older flies is not due to blockage in the outward movement of Rho. The results suggest that after localizing within the rhabdomeres, the inability of Rho to interact correctly with cellular structural components precipitates the unraveling of the rhabdomeres. In this view, the rhabdomeric atrophy previously observed in Rh1 rhodopsin null alleles (1) is not simply due to the lack of protein populating the rhabdomeric membranes. Rather, Rh1 rhodopsin contains motifs, not present in Rho, that are able to

Figure 2. Expression and localization of Rh1-Rho chimeras. Schematic representation of the secondary structure, protein expression, localization, and amino acid alignment of rhodopsin chimera RhoC2-Rh1 (A–D), RhoC3-Rh1 (E–H), RhoCt-Rh1 (I–L). The alignment of amino acid residues contributed by Rh1 (shaded circles) and Rho (open circles) is limited to the residues present in the loops used in chimera construction. The numbering of residues flanking the alignment is based on the primary sequence of rhodopsin. Identical residues are highlighted by solid boxes; conservative substitutions of residues are highlighted by open boxes, where I=V, F=Y, S=T. Protein blots of head homogenates expressing rhodopsin chimeras show comparable expression levels with the control flies. Immunostaining of the LRW embedded retinal sections show localization of chimera proteins in the R1–6 rhabdomeres with no detectable signal in R7 cell (arrow). For RhoCt-Rh1 chimera, Rho-expressing flies were used as controls because the epitopes for anti-Rh1 and anti-Rh4 Ab are present on the cytoplasmic tail (Ct) domain.
interact with other structural components to specify rhabdomere integrity.

The rhodopsin cytoplasmic loop domains are positioned for interaction with cytoskeleton and signaling components of the cell. To investigate the possibility that a determinant on the cytoplasmic loops C2, C3, and Ct of Rh1 opsin is required for rhabdomeric structure, we substituted each domain individually with the corresponding Rho loops. The cytoplasmic loop (C1) was not included in the analysis because structural analysis shows it does not significantly project out of the membrane (18, 19) and has good conservation of amino acid residues (6 of the 12 residues are identical) between Rho and Rh1 (4, 5). For these two reasons, it is unlikely that the C1 loop could specify the differences between Rh1 and Rho behavior.

In transgenic flies carrying substitution of Rh1 cytoplasmic loops C2 and C3 individually with the corresponding Rho loops, the rhabdomeric size and integrity remained similar to native Rh1, thereby ruling out the presence of any unique determinants on these loops essential in the maintenance of rhabdomeric morphology. However, the substitution of Rh1 Ct domain with the Rho Ct domain resulted in smaller and irregularly shaped rhabdomeres at 7 and 14 days. Further, a substantial improvement of Rho photoreceptors’ disorganized rhabdomeres is seen in a rhodopsin chimera that contains the Rh1 Ct domain on the otherwise complete Rho protein. These results establish that the *Drosophila* Rh1 cytoplasmic tail domain plays a key role in organizing rhabdomere structure.

The cytoplasmic tail of vertebrate rhodopsin plays a role in Rho trafficking to the rod outer segment (20–23). In *Drosophila* photoreceptors, both Rho and the Rhoc3-Rh1 chimeric proteins localize to rhabdomeres. No rhodopsin accumulation was observed within the endoplasmic reticulum or basolateral membranes as expected for defective trafficking processes. This suggests that motifs promoting trafficking to rhabdomeres in *Drosophila* photoreceptors are found on both Rho and Rh1. Rather, the distinction between Rho and Rh1 is that only Rh1 retains motifs promoting maintenance of rhabdomeric membrane, presumably as a result of rhodopsin-rhodopsin contacts and association with other intracellular cytoskeleton components. An actin cytoskeletal network organizes microvillar structures, including the rhabdomere (1). Within this environment, the unconventional myosin NINAC (24, 25) acts to stabilize the microvillar structure of the *Drosophila* photoreceptor rhabdomeres, but no other molecular components involved in rhabdomere maintenance are well characterized.

Rhodopsin is the major protein of the rhabdomere, representing greater than 50% of the protein content (26). Structural information has been reported for only one invertebrate rhodopsin, the squid rhodopsin. While the overall folding of this invertebrate rhodopsin is similar to vertebrate, the carboxyl-terminal domain contains novel structural domains. The complete carboxyl-terminal domain contains a proline-rich segment that promotes clustering of rhodopsin (27). When only this proline-rich segment is removed, an alpha-helical domain promoting linear assembly of rhodopsin molecules is revealed (28). This segment is thought to interact with the expanded 3rd cytosolic loop found on all invertebrate rhodopsins. Our analysis of *Drosophila* rhodopsin, which lacks the proline-rich domain, confirms a structural role of an invertebrate rhodopsin carboxyl-terminal domain under *in vivo* conditions. There is low sequence identity of *Drosophila* and squid in this region, so additional analysis will be required to compare and contrast the role of structural motifs. Also, our results have not implicated the expanded 3rd cytoplasmic loop of the invertebrate rhodopsins, as substitution of the *Drosophila* 3rd cytoplasmic loop did not notably impact rhabdomere morphology.

Placing the Rho carboxyl-terminal domain on Rh1-affected rhabdomere morphology but did not mimic the extensive rhabdomeric membrane unraveling phenotype originally observed in Rho flies. Further, replacement of the carboxyl-terminal domain of Rho with the corresponding region of Rh1 dramatically improved rhabdomere structure but still exhibited some membrane unraveling at 14 days of age. It is possible the complete Rho phenotype is due to the simultaneous loss of the Rh1 carboxy tail and additional Rh1.
The Rh1 transmembrane helices could also play a role in rhabdomeric integrity. In vertebrate rod outer segment disks, regions of transmembrane helices close to the extracellular surface form weak interactions across the rows of rhodopsin embedded in membrane disks (18, 19). Similar interactions between Rh1 transmembrane helices could contribute to maintaining rhabdomeric integrity. Retinal chromophore binding is also critical to rhodopsin conformation. We showed that dark-reared Rho expressing flies show similar rhabdomeric unraveling as light-reared flies. These results discount the possibility that light-induced conformation change in Rho due to chromophore isomerization or bleaching is creating a Rho intermediate that disrupts rhabdomere structure. A second consideration is that Rho expressed in Drosophila is complexed with 3-OH retinal instead of retinal (15), and this configuration may contribute to structural instability.

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REFERENCES


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