

**E07-10-1070 Yoder**

**Supplementary Figure Legends**

**Supplementary Figure 1.** Expression of *xbx-7*, *tza-1*, and *tza-2* in *C. elegans*. (A) *xbx-7::YFP* and *tza-1::CFP* and (B) *tza-2::CFP* and *xbx-7::YFP* transgenes were expressed in an overlapping pattern the ciliated sensory neurons of the worm including the (left) amphid and labial neurons in the head as well as the (right) phasmid neurons in the tail.

**Supplementary Figure 2.** Sequencing of the *tm2705*, *tm2452*, and *ok2092* mutant transcripts. RNA isolated from mutant worms was reverse transcribed, amplified by PCR, and then sequenced. Nucleotide numbering corresponds to the position in the wild-type gene. (A) The *xbx-7(tm2705)* deletion causes the resulting transcript to splice exon 2 (ending at nucleotide 535) with exon 5 (beginning at nucleotide 1724). The original reading frame (solid vertical lines) is not altered by this mutation. (B) The *tza-1(tm2452)* deletion results in a transcript that splices exon 1 with a cryptic splice acceptor in exon 3 created by the fusion of nucleotides 445 and 725. The original reading frame is not altered by this mutation. (C) The *tza-2(ok2092)* deletion fuses nucleotide 573 in exon 1 with nucleotide 1381 in exon 4. This causes a shift of reading frame (dashed lines) in the resulting transcript.

**Supplementary Figure 3.** Yeast two-hybrid analysis of possible interactions between the B9 proteins. Yeast expression constructs were generated containing full length XBx-7, TZA-1, or TZA-2 cDNA and cotransformed into yeast. (Top row) Low stringency selective media lacking

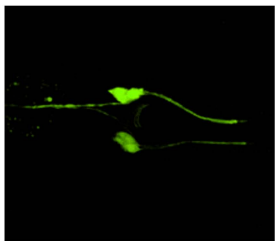
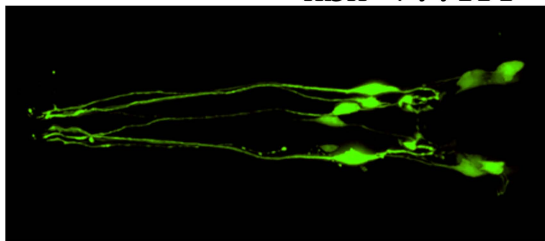
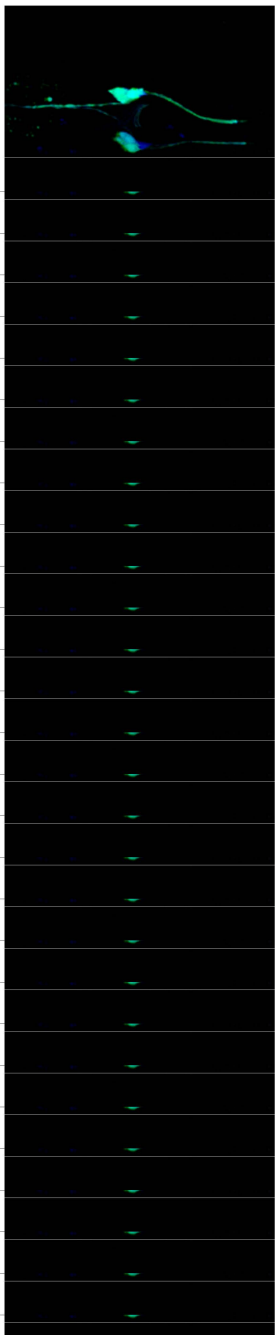
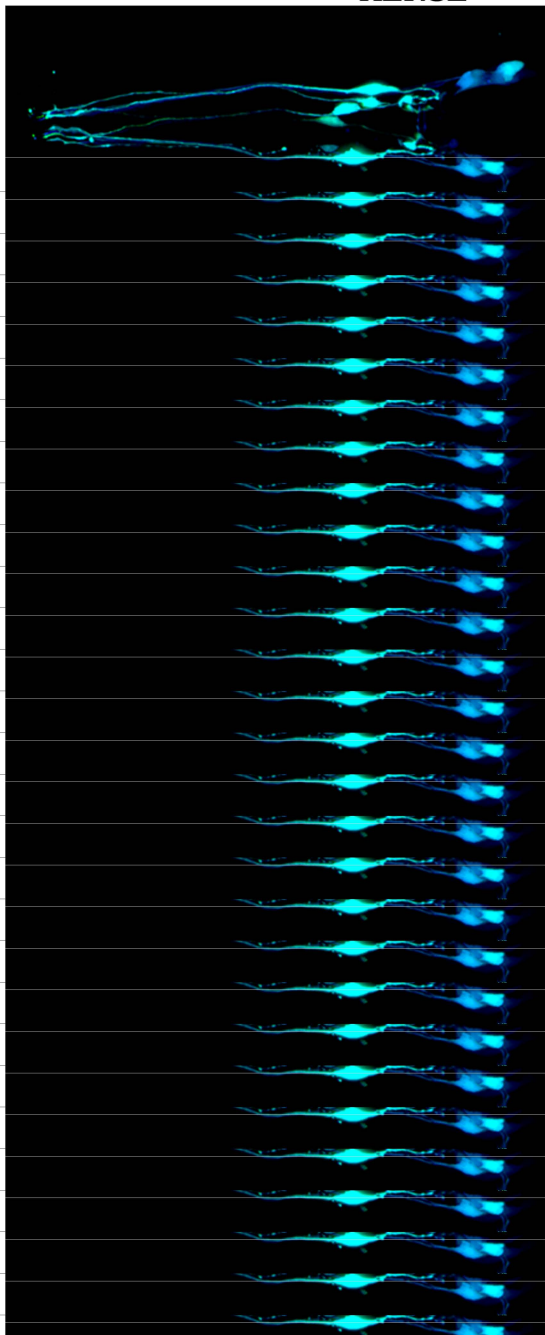
B9 proteins, NPH-4, and cilia morphology

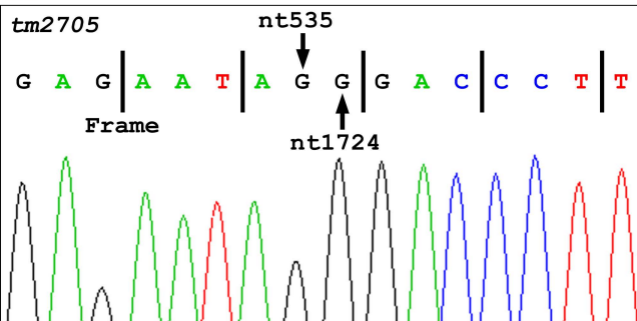
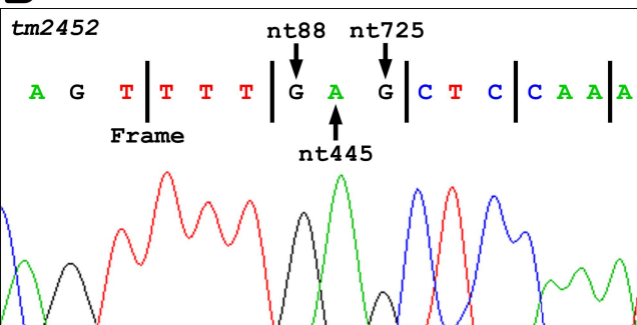
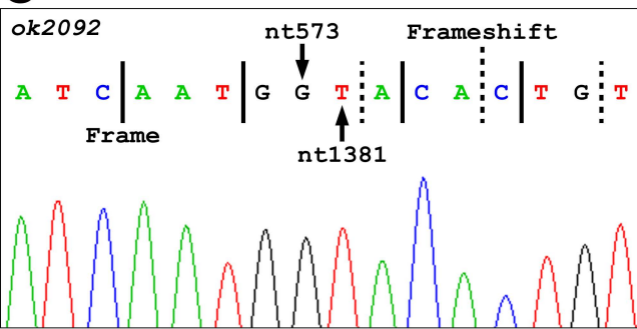
Trp and Leu was used to isolate yeast colonies containing both cotransformed constructs.

(Bottom row) Colonies expressing the fusion proteins were then tested for possible interactions on high stringency selective media containing X- $\alpha$ -Gal and lacking Trp, Leu, His, and Ade.

(First column) The positive controls p53 and large T-Antigen grew on selective media and expressed the  $\alpha$ -galactosidase reporter gene. (Second column) Yeast strains cotransformed with XBX-7 and TZA-1 grew on low stringency selective media but failed to grow when plated on high stringency selective media, suggesting that XBX-7 does not physically bind TZA-1. (Third column) Yeast strains cotransformed with XBX-7 and TZA-2 grew on low stringency selective media but failed to grow when plated on high stringency selective media, suggesting that XBX-7 does not physically bind TZA-2. (Fourth column) Yeast strains cotransformed with TZA-1 and TZA-2 grew on low stringency selective media and also grew when plated on high stringency selective media and expressed the  $\alpha$ -galactosidase reporter gene, confirming the positive interaction between TZA-1 and TZA-2 reported previously (Li *et al.*, 2004).

**Supplementary Figure 4.** NPH-1 and NPH-4 localization is not affected by mutations in the B9 genes. To analyze the effect of *xbx-7(tm2705)*, *tza-1(tm2452)*, or *tza-2(ok2092)* mutations on NPH-1::CFP or NPH-4::YFP localization, strains were generated by passing extrachromosomal arrays encoding NPH-1::CFP or NPH-4::YFP from wild-type worms into the mutant backgrounds. The cilia/transition zone region of one amphid cilia bundle is shown in each panel. Compared to wild type, (top) NPH-1::CFP localization and (bottom) NPH-4::YFP localization to the transition zones of amphid neurons was not altered in the *xbx-7(tm2705)*, *tza-1(tm2452)*, or *tza-2(ok2092)* mutant backgrounds.

**A***xbx-7::YFP***MERGE**

**A****B****C**

**Selection**

-Trp -Leu

-Trp -Leu

-His -Ade

+X- $\alpha$ -Gal

p53  
T-antigen

XBX-7  
TZA-1

XBX-7  
TZA-2

TZA-1  
TZA-2

