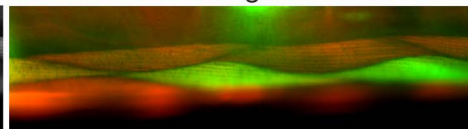
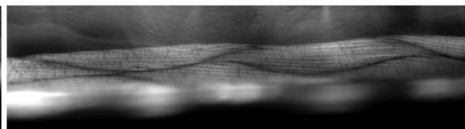
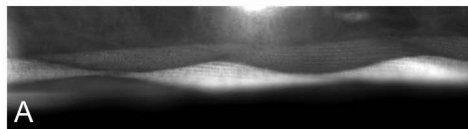


GFP-UNC-78

Actin

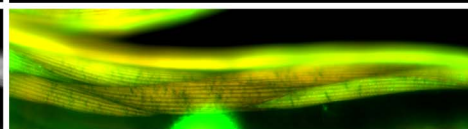
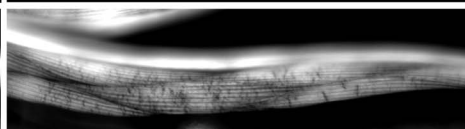
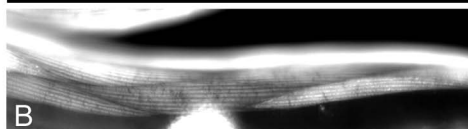
Merged

WT



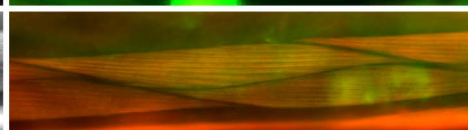
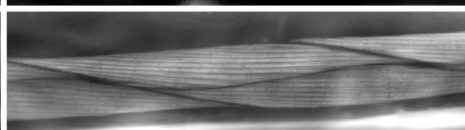
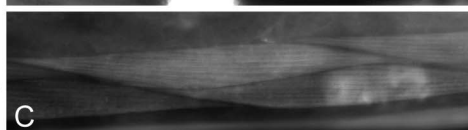
A

E126A



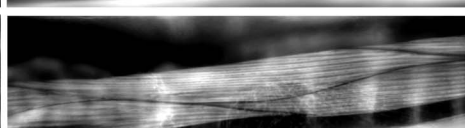
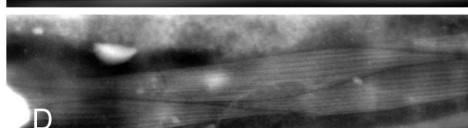
B

D168A



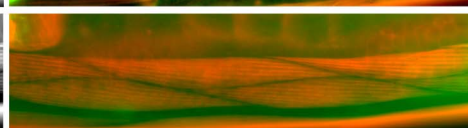
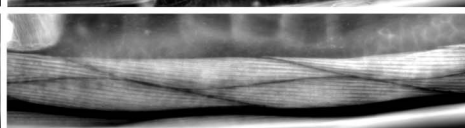
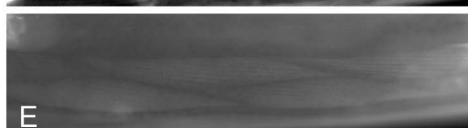
C

K181A



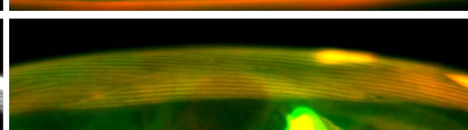
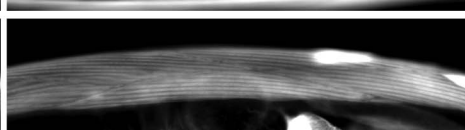
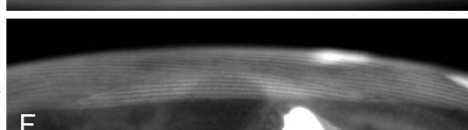
D

F182A



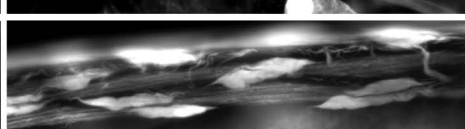
E

F192A

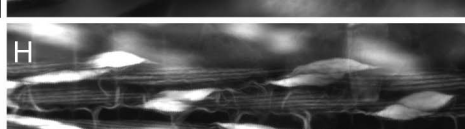


F

4x



G

unc-78(null)

H

20μm

Supplemental Figure 1. GFP-UNC-78 with a quadruple mutation (E126A, D168A, F182A, and F192A) fails to rescue defects in the actin organization in the *unc-78(null)* mutant. Adult *unc-78(null)* worms that express GFP-UNC-78 were stained by tetramethylrhodamine-phalloidin to visualize organization of the actin filaments in the body wall muscle. Wild-type (A) or single mutants (B-F) rescued the striated organization of the actin filaments, while the 4X mutant did not (G). The *unc-78(null)* mutant without transgenic expression is shown in H. Bar, 20 μm .