Fig. S1, related to Fig. 1

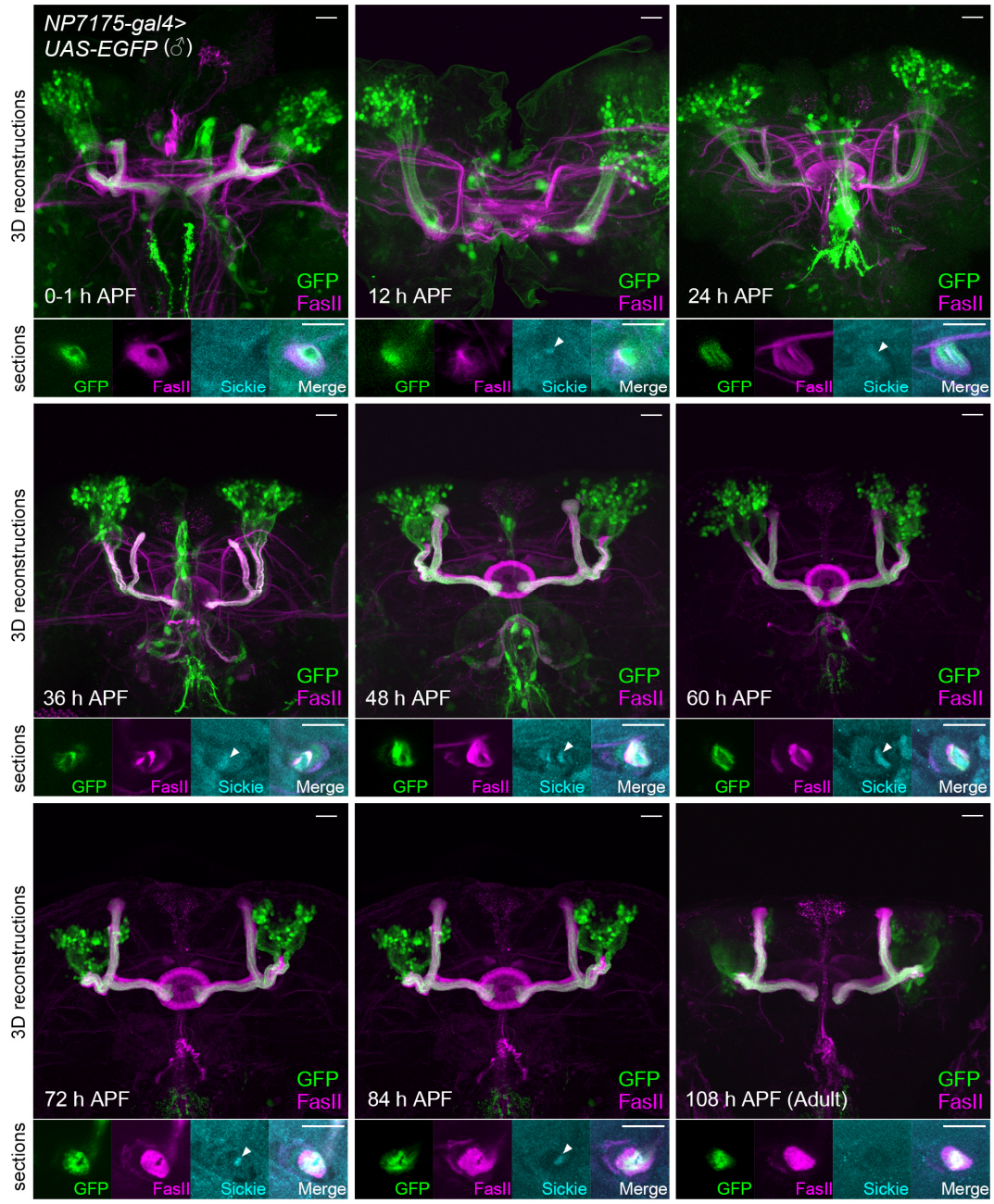

Fig. S1. The expression pattern of *NP7175-gal4* at pupal stage.

Throughout development, GFP signals were detected in the inner regions of FasII-labeled α/β axonal bundles. From 12 to 84 h APF, Sickie expression was prominently detected in the core regions of the axonal bundles (arrowheads) in which FasII signals were weak. We did not detect strong GFP signals in this exact core region of the peduncle. This result might be due to a delay in the Gal4/UAS system. Scale bar, 20 μm.

Fig. S2., related to Fig.5

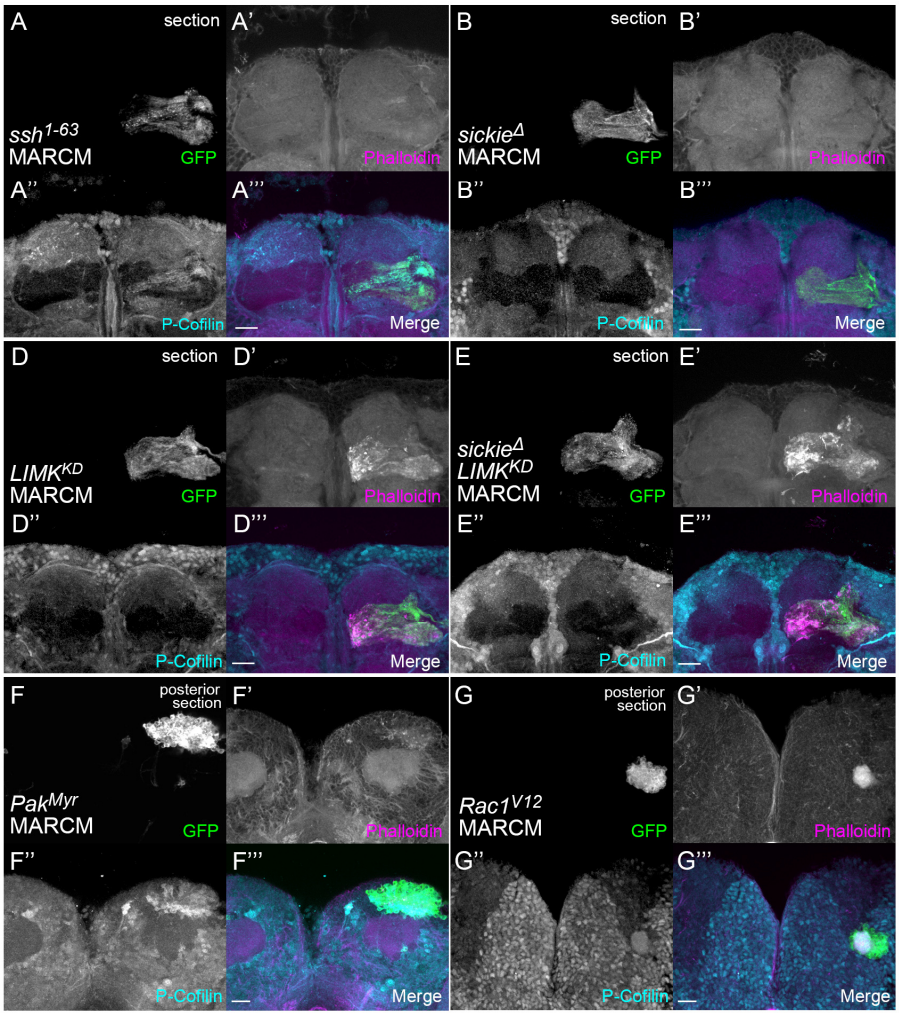

Fig. S2. Duplicated Fig. 5 images free of markings.

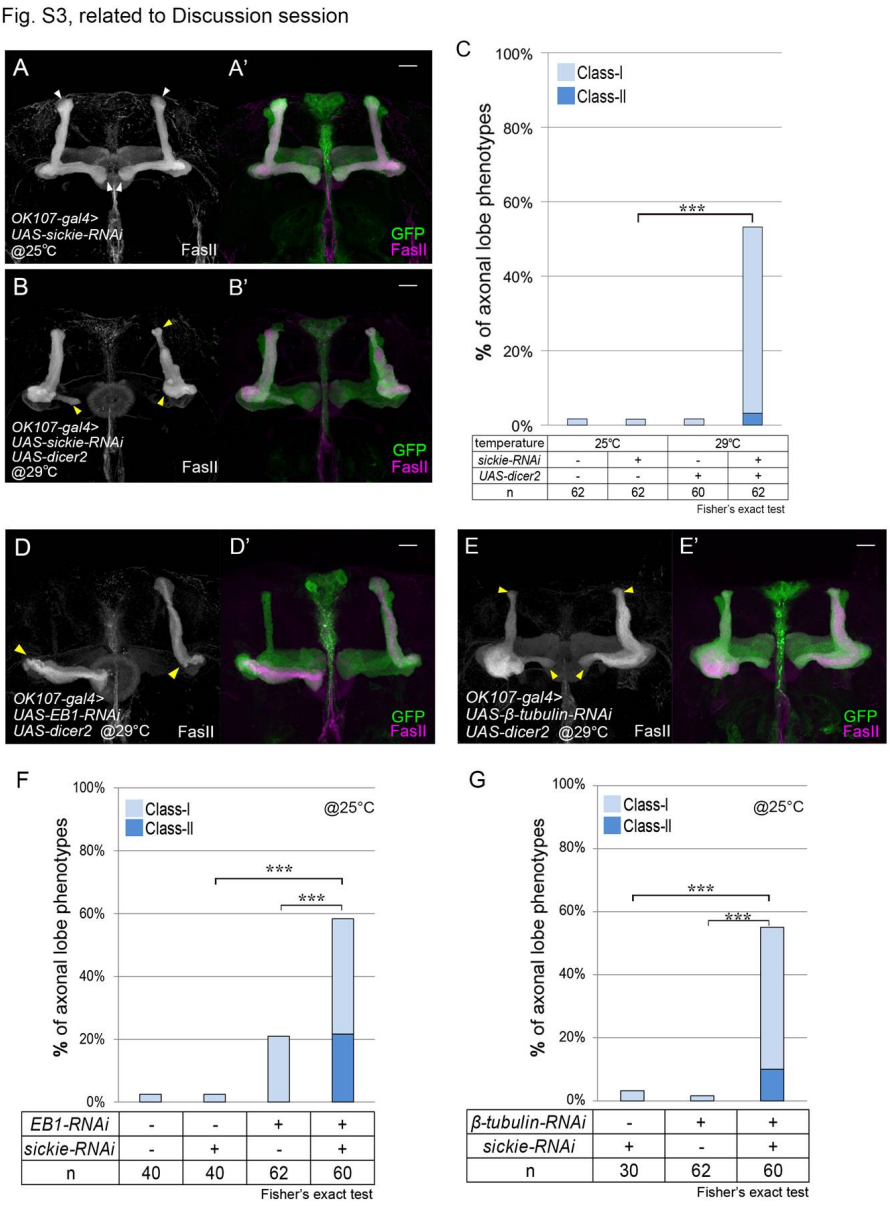

Fig. S3. Sickie genetically interacts with microtubule components.

(A-A') Any obvious axonal growth defect was not detected when *sickie-RNAi* knockdown was singly induced at 25°C. (B-B') Some RNAi-treated flies show axonal defects when RNAi was induced with *dicer-2* co-expression at 29°C. (C) The penetrance of the axonal defect was significantly increased when *dicer-2* was co-expressed and the flies were reared at 29°C. ***$p=1.50\times10^{-11}$. (D-D',E-E') RNAi of either *EB1* or *β-tubulin* induced lobe

formation defects with *dicer-2* co-expression at 29°C. (F,G) Either *EB1-* or *β-tubulin-RNAi* also showed low penetrance of the axonal lobe formation defects at 25°C. However, compared with the single knockdown of *sickie*, the penetrance of the axonal defects synergistically increased in both RNAi treatments by combining with *sickie-RNAi,* even at 25°C. *sickie/sickie-EB1-RNAi*: ***p=1.54×10^{-9}, *EB1/sickie-EB1-RNAi*: ***p=3.63×10^{-5}, *sickie/sickie-β-tubulin-RNAi*: ***p=4.88×10^{-7}, and *β-tubulin/sickie-β-tubulin-RNAi*: ***p=3.25×10^{-12}. Scale bar, 20 μm.