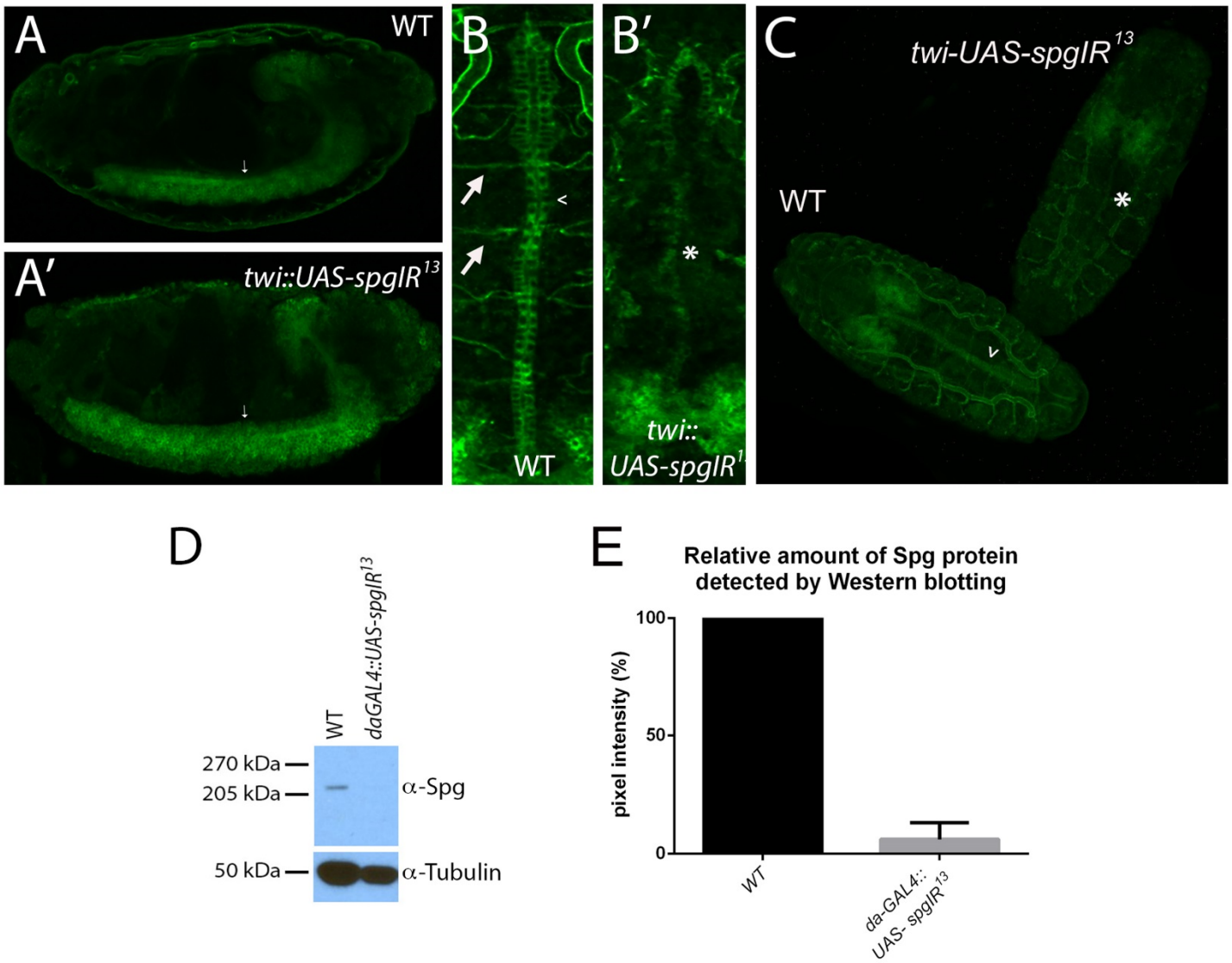

Figure S1 Spg protein expression is decreased using RNAi knockdown. (A-C) Fluorescently labeled stage 17 embryos labeled with an antibody against the Spg protein. (A, A') *WT* and *twi::UAS-spgIR*^{13} embryos both show Spg protein expression in the developing central nervous system (arrows), as the *twi-GAL4* driver is not active in the nervous system. (B-C) Dorsal views of stage 17 embryos stained for anti-Spg protein. (B) Spg protein is normally detected in cardioblast cells (carat) and alary muscles (arrow) in *WT* embryos. (B') Spg expression is decreased in the cardioblasts (asterisk) upon knockdown of *spg* using RNAi driven with the *twi* promoter. (C) Spg is apparent in the dv of *WT* (carat), but not *twi::UAS-spgIR*^{13} embryos (asterisk). Image was taken of both embryos together to eliminate variations in data collection. (D) *WT* or *spg RNAi* embryos were selected and subjected to Western blotting for anti-Spg (top panel) or the loading control anti-tubulin (bottom panel). Spg is apparent at the expected molecular weight in WT samples (left lane), but is absent upon knockdown of *spg* using the ubiquitious *daughterless-GAL4 (da-GAL4)* driver (right lane). (E) Quantitation of relative pixel intensity of the Western blot in D. Values are normalized to the loading control and results are the average of two independent experiments.

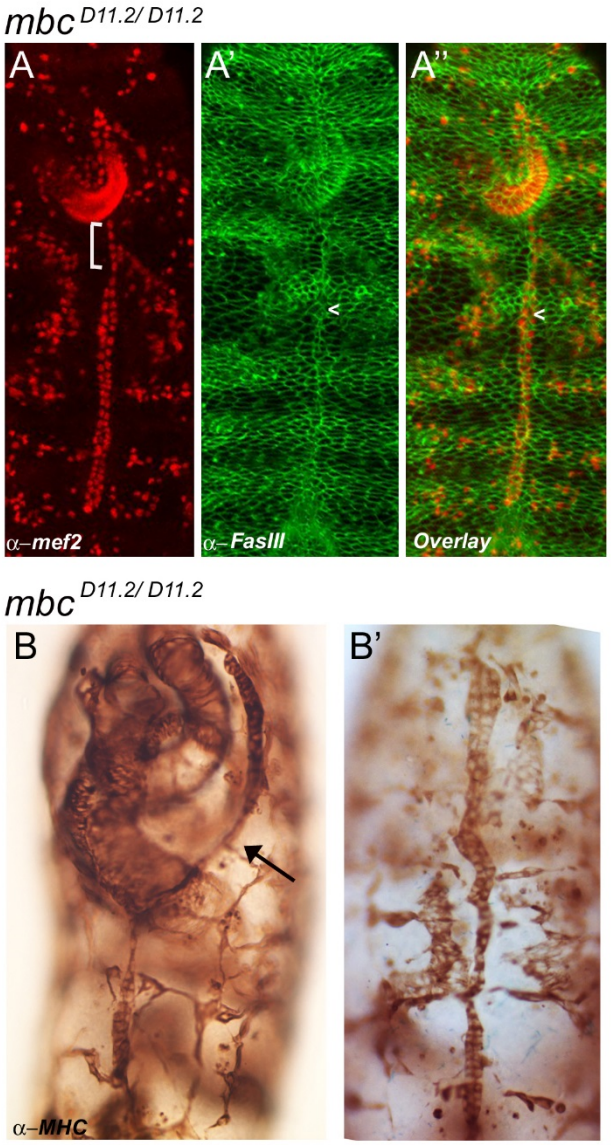

Figure S2 Dorsal closure in mutants with defective dorsal vessel development. (A-B') St. 17 mutant embryos stained fluorescently (A-A") or colorimetrically (B, B') to view the dorsal vessel and overlying epidermis. (A-B') *mbc*$^{D11.2/D11.2}$ mutant embryos to examine dorsal closure and dorsal vessel defects. The majority of embryos (90%) do not exhibit dorsal closure defects as visualized by FasIII (green), but still exhibit defects in dv patterning (Mef2 in red; bracket). (B) Approximately 10% of *mbc* mutants show dorsal closure defects. If this occurred, the cardioblasts did not migrate to the midline (arrow; compare to the midline pairing of cardioblasts in B'). Embryos that showed dorsal closure defects were not included in our phenotypic or quantitative analysis of dv phenotypes.