Figure S1

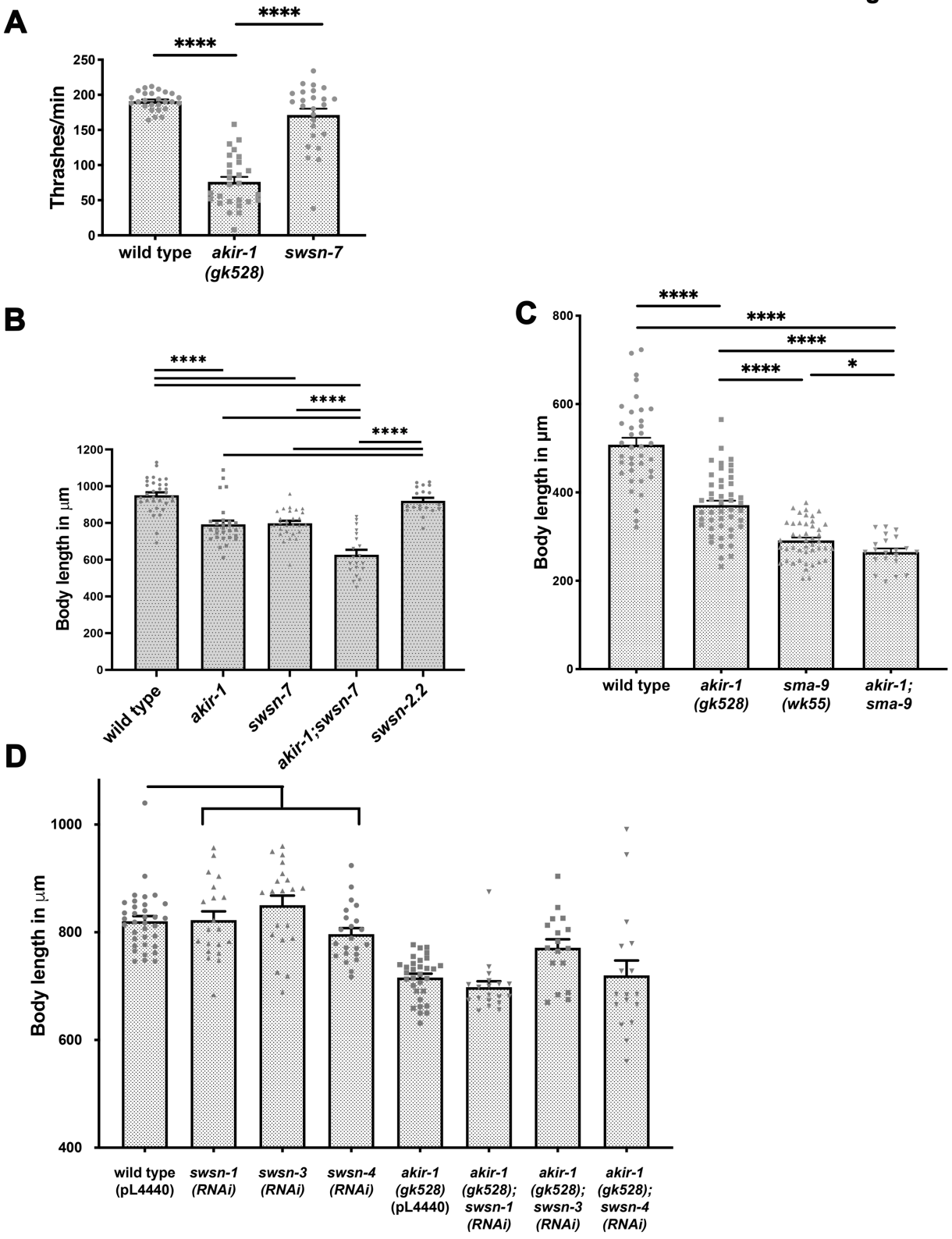

Figure S1: AKIR-1 does not genetically interact with genes in the SWI/SNF pathways.

A) *swsn-7* mutants do not show defects in movements. B) *swsn-7* mutants show reduced body size, but these defects are additive when combined with *akir-1* indicating that *akir-1* and *swsn-7* act in different pathways. wild type n=32, *akir-1* n=28, *swsn-7* n=27, *akir-1;swsn-7* n=20, *swsn-2.2* n=19. C) Body lengths of wild type n=37, *akir-1(gk528)* n=47, *sma-9(wk55)* n=45, *akir-1(gk528);sma-9(wk55)* n=21 scored at the L3 developmental stage D) Body lengths of wild type and *akir-1(gk528)* after exposure to RNAi targeting the indicated gene from L1 to Day 1 Adult. RNAi penetrance is incomplete as indicated by surviving animals (null alleles of these genes are embryonic or larval lethal), and as such the data is inconclusive. * $0.001 \leq p < 0.05$*** $0.0001 \leq p < 0.001$**** $0.0001 \geq p$.

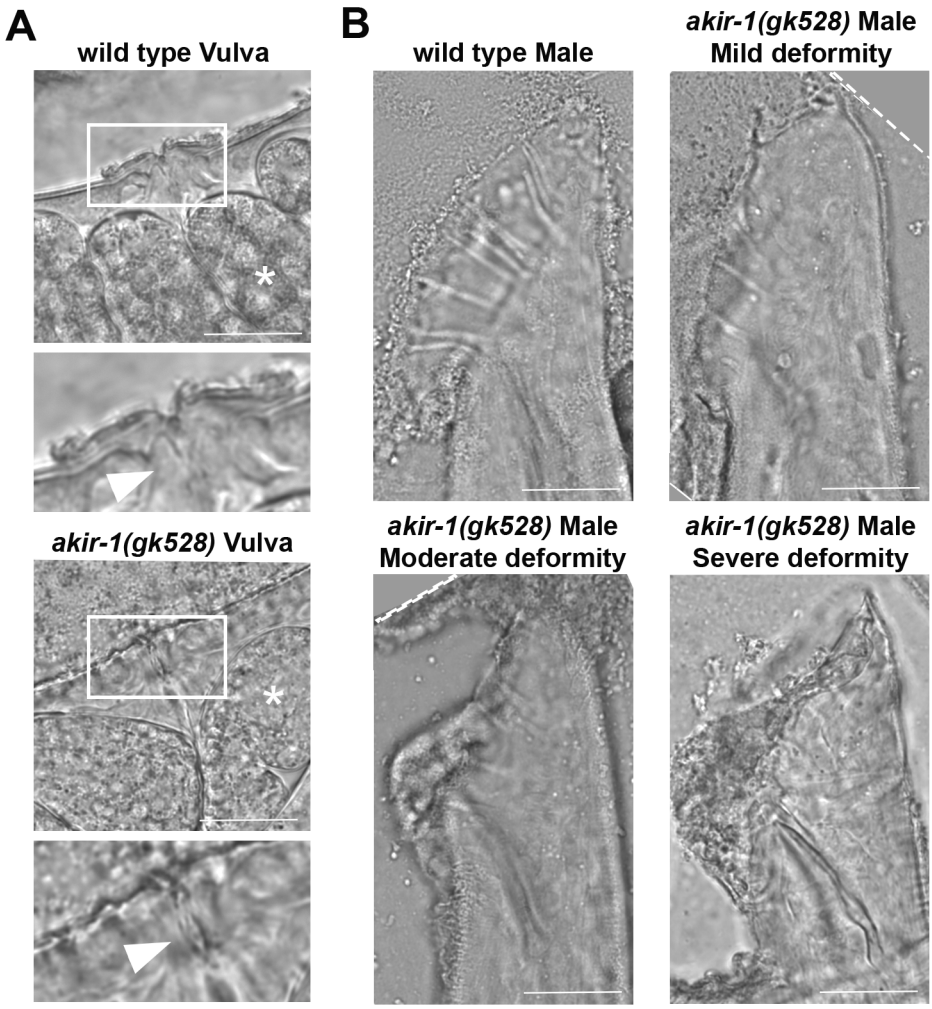

Figure S2: AKIR-1 shows developmental defects of tail and vulva structures.

Representative DIC images of morphology of the vulva (A) and tail (B) in wild type and *akir-1* mutants. A) *akir-1(gk528)* mutants display vulvar defects likely stemming from lack of full muscle development (M-lineage defects Figure 3CD). The image shows wild type vulva with normal muscle formation below the cuticle (arrowhead), slightly protruding outward with visible opening. In the *akir-1(gk528)* mutant you can see the defects in muscle development resulting in a lack of vulvar protrusion. This leads to inability to efficiently lay eggs, with embryos at more

advanced stages of development remaining in the animal uterus (asterisk). B) Representative images of the severity of *akir-1(gk528)* male tail defects (also see figure 6A). Scale bar 25μm.