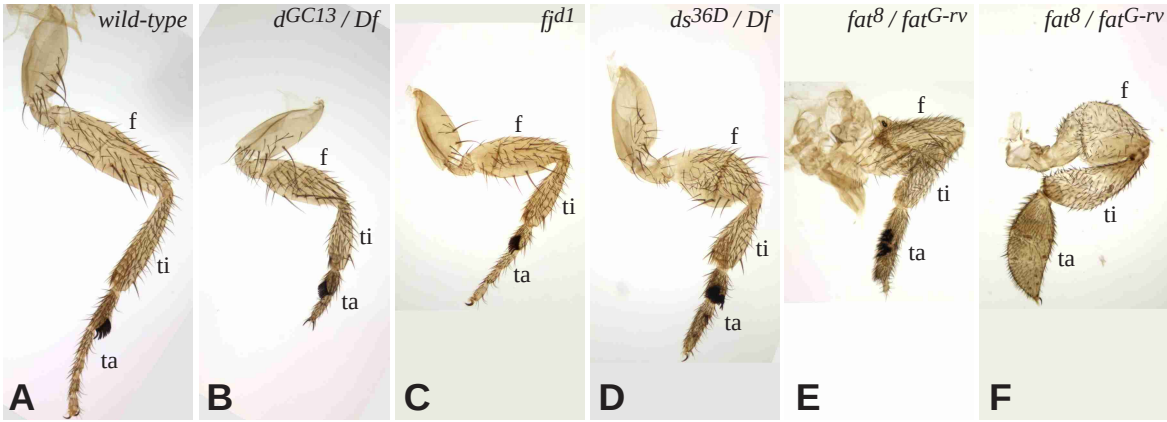
Supplementary Fig. S1



Adult legs, all at the same magnification, from A) *Wildtype* B) d^{GC13} / *Df(2L)ED623* C) fj^{d1} D) ds^{36D}/ *Df(2L)ED94* E,F) fat^{8}/ $fat^{G\text{-}rv}$. The femur (f), tibia (ti) and tarsus (ta) are labeled. Mutations in each of these Fat pathway components result in shortened legs, but their phenotypes are distinct. *fj* and *dachs* legs are simply shortened, *dachs* more so than *fj*. *ds* mutant legs are shortened but also thicker. *fat* mutant legs are shorter and sometimes much thicker. The *fat* phenotype is more variable (two examples are shown). There are at least three distinct processes affecting leg development that could be impaired by these mutants. First, there are autonomous influences of Fat on growth. Second, there are affects on the expression of Notch ligands, which also influence growth. Third, there might be affects on the orientation of cell divisions, as has been described in the wing. The observation that *fj* and *ds* have weaker phenotypes than *fat* implies that some Fat "activity" is independent of these Fat regulators.