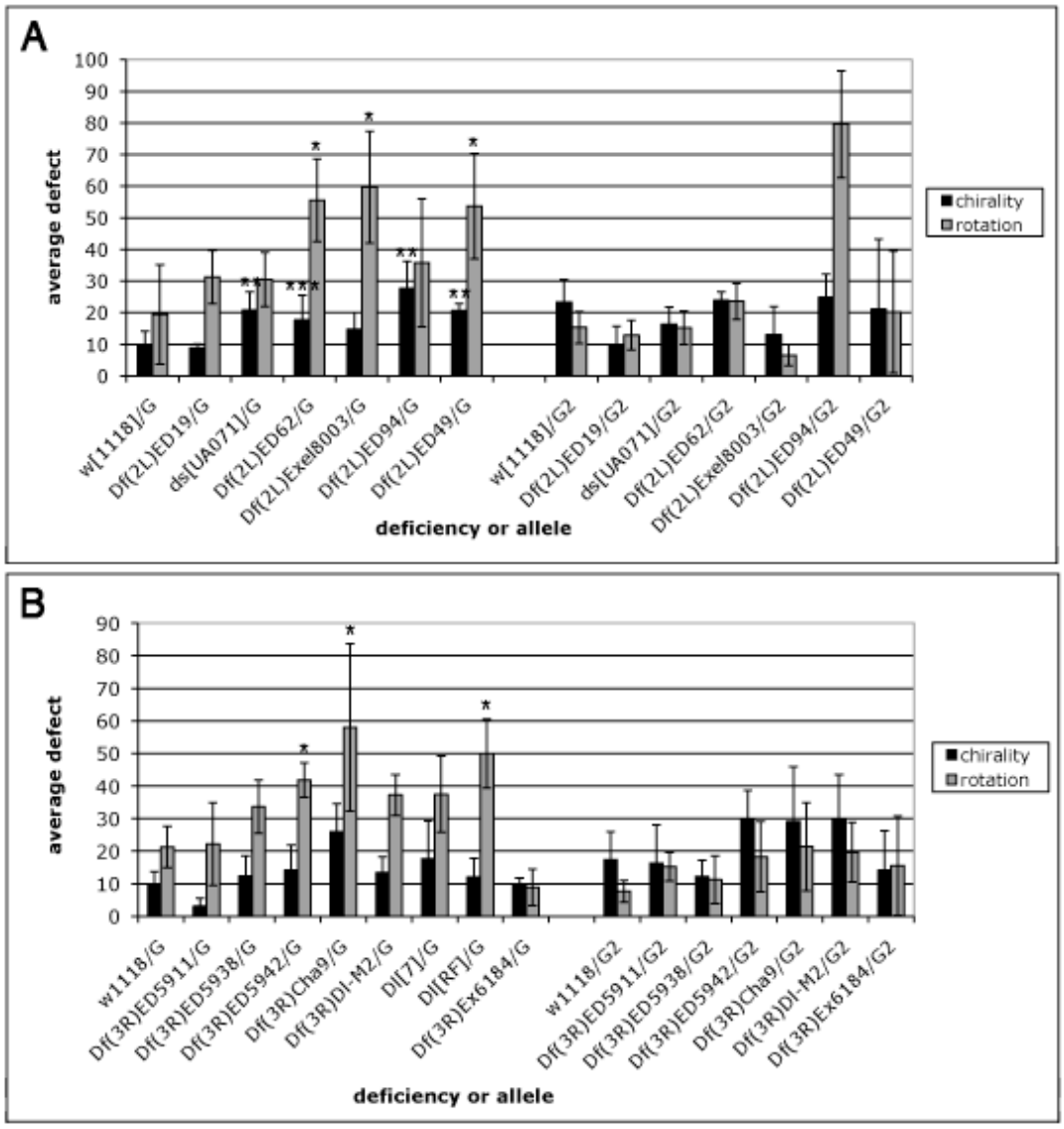

Figure S1 Two interacting DrosDel deficiencies identified in the initial screen covered two known factors, *ds* and *Dl*, acting in parallel to or downstream of the Fz/PCP signaling pathway. Graphs show average rotation and chirality defects as determined by the *Rh1-GFP* assay for indicated genotypes for both *sev-Gal4*, *UAS-dgo* (G) and *sev-Gal4*, *UAS-pk* (G2). (A) *Df(2R)ED62*, subdividing deficiencies *Df(2R)ED49* and *Df(2R)Exel8003* enhanced rotation defects of *sev-Gal4, UAS-dgo* significantly (*=P<0.03). *Notchless* (*Nle*) might be the candidate gene responsible for that interaction. Subdividing deficiencies *Df(2R)ED49, Df(2R)ED94* and ds^{UA071} enhanced chirality defects of *sev-Gal4, UAS-dgo* significantly (**=P<0.02), confirming the initial chirality interaction (***=P<0.1) and identifiying *dachsous(ds)* as the gene responsible for it. No effects were seen with *sev-Gal4*, *UAS-pk*. *Df(2R)ED94* enhanced rotation defects of *sev-Gal4, UAS-pk.* (B) *Df(3R)ED5942*, subdividing deficiencies *Df(3R)Cha9* and Dl^{RF} enhanced rotation defects of *sev-Gal4, UAS-dgo* significantly (*=P<0.1), confirming the initial interaction and identifying *Delta(Dl)* as the gene responsible for it. Deficiency *Df(3R)Cha9* also enhanced chirality defects of *sev-Gal4, UAS-dgo.* 4 eyes were analyzed each and 90-150 ommatidia were evaluated per genotype.