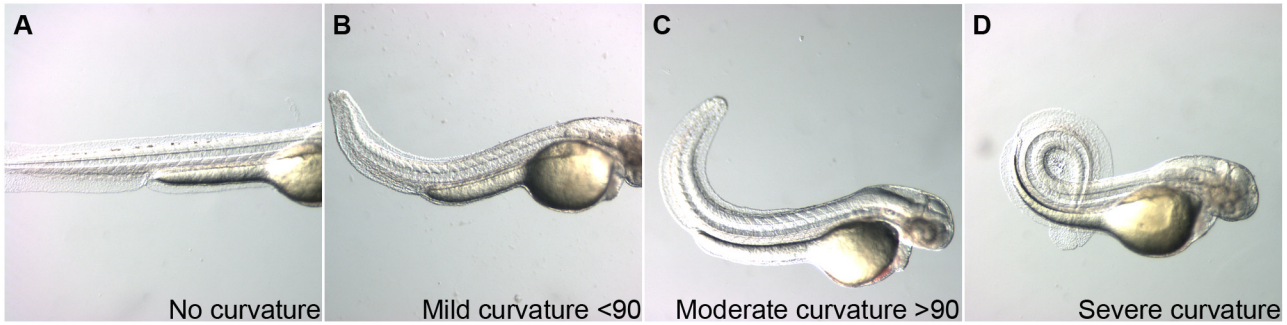
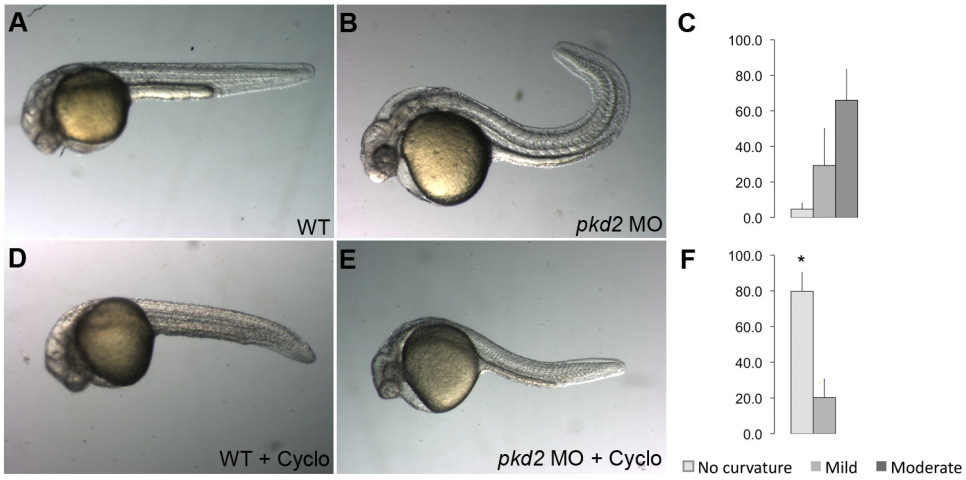
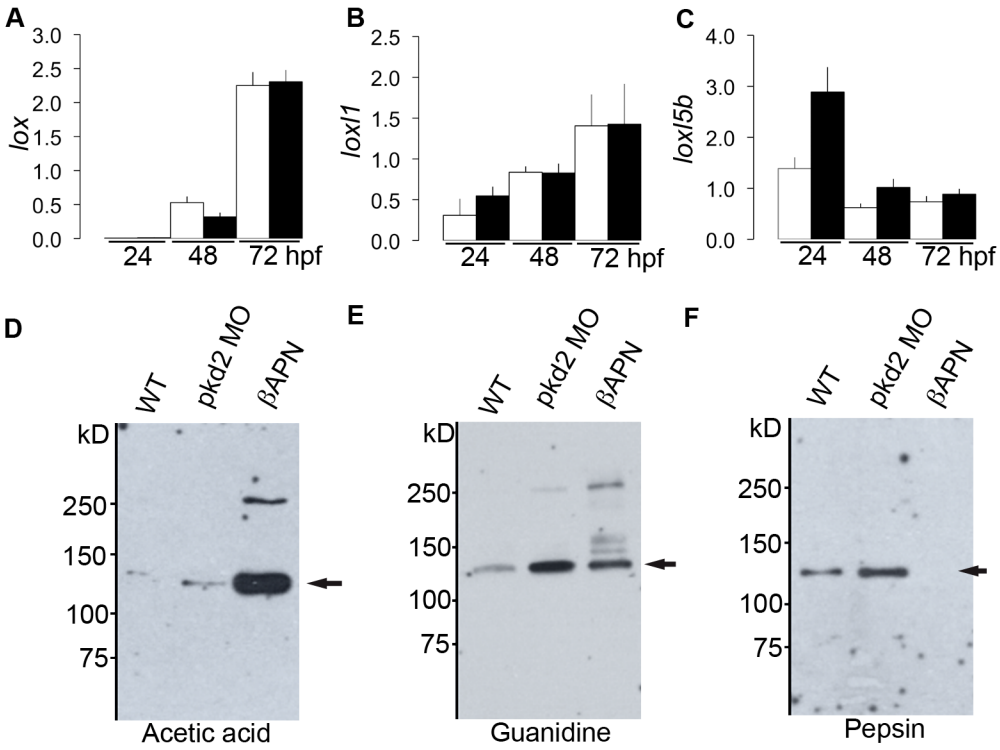


Supplementary figure 1: Range of severity in dorsal axis curvature. We used a range of severity in dorsal axis curvature to score embryos at 48 hpf and considered it as straight when there was no curvature (A), mild when it was less than 90 degrees (B), moderate when it was more than 90 degrees (C) and severe when the tail was crossing the body axis (D).

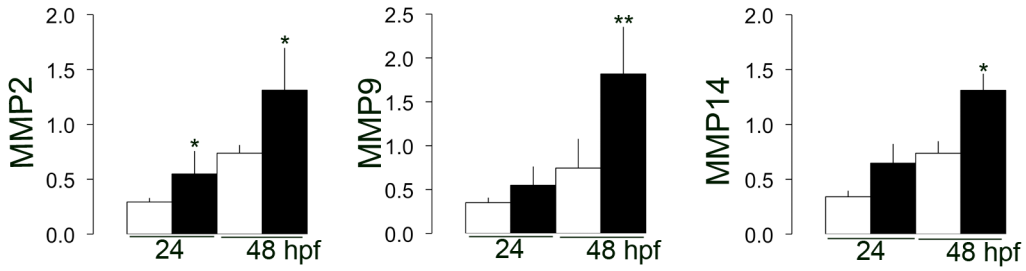
Supplementary figure 2: (A-C) *col2a1a* morphants, *double bubble* embryo (C) and *col2a1a* knock-down in *double bubble* mutant (C) at 96 hpf, showing no rescue of the ventral curvature.



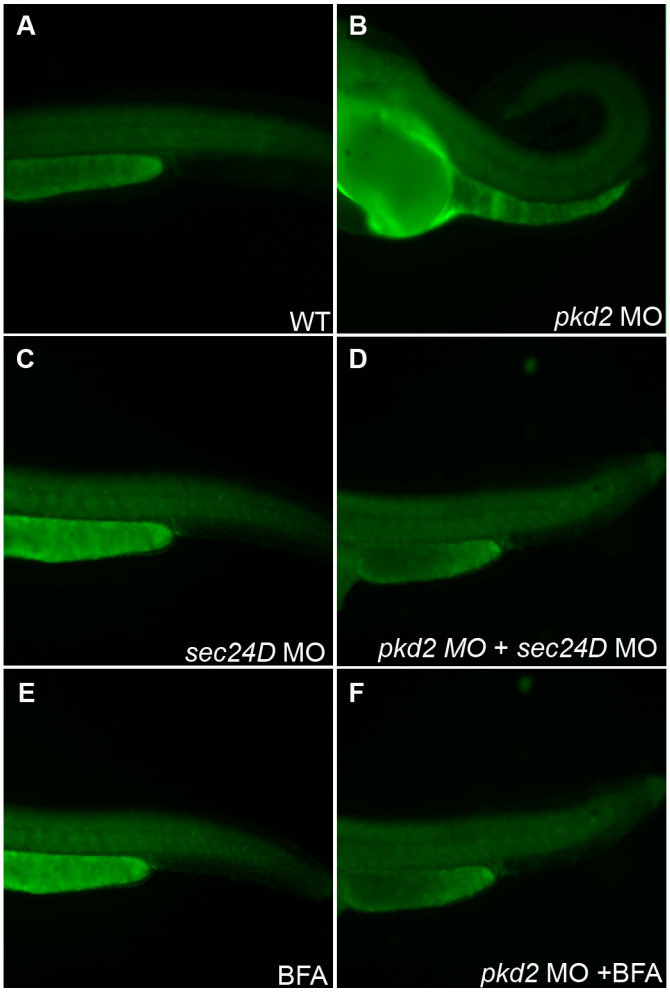
Supplementary figure 3. Low dose cycloheximide is sufficient to rescue *pkd2* morphant axis curvature. (A) Control 30 hpf embryo. (B) *pkd2* morphant at 30 hpf showing moderate axis curvature. (C) Quantification of the number of *pkd2* morphant embryos showing moderate, mild or no curvature. (D) Control cycloheximide (20 µg/ml) treated 30 hpf embryo. (E) 30 hpf *pkd2* morphant embryo treated with 20 µg/ml cycloheximide starting at 24 hpf. (F) Quantification of number of cycloheximide treated *pkd2* morphant embryos showing moderate, mild or no curvature. Most cycloheximide treated embryos showed no curvature (*; p=0.02)



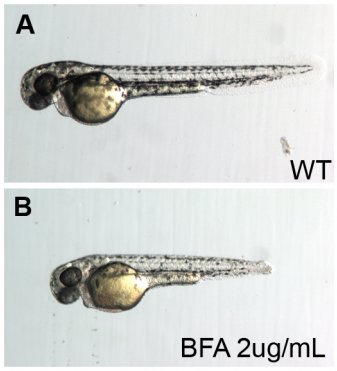
Supplementary figure 4: Collagen type II cross-linking is not affected in *pkd2*-deficient embryos. (A-C) Quantitative RT-PCR measurements of *lox* (A), *loxl1* (B) and *loxl5b* (C) showing no difference in their expression between wildtype embryos (white bars) and *pkd2* morphants (black bars) at 24, 48 and 72 hpf. (D-F) SDS-PAGE analysis of collagen type II serially extracted from wildtype embryos, *pkd2* morphants and bAPN-treated embryos by 3% acetic acid (D), 4M guanidine-HCl (E) and pepsin digestion (F) at 48 hpf. bAPN-treated embryos mostly have soluble collagen type II while most is cross-linked in wildtype embryos and *pkd2* morphants based on the relative extractabilities. Arrows indicate the size of α1 chains of collagen type II.



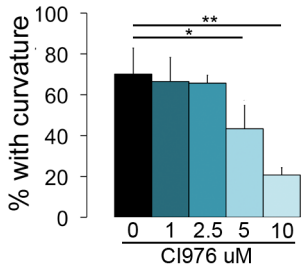
Supplementary figure 5: Collagen accumulation is not due to a lack of mmp gene expression. Quantitative RT-PCR measurements of *mmp2* (A), *mmp9* (B) and *mmp14* (C) showing an increased expression in *pkd2* morphants (black bars) compared to wildtype embryos (white bars) at 48 hpf. n = 4 per group.



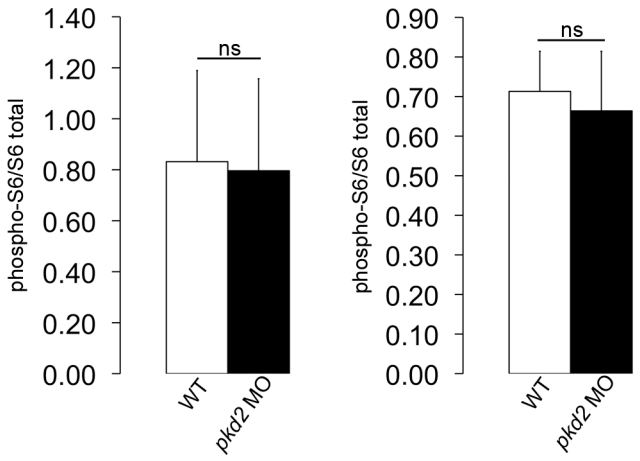
Supplementary figure 6: *sec24D* knock-down and BFA treatment do not induce apoptosis. (A-F) Acridine orange staining in wildtype embryos (A), *pkd2* morphants (B), *sec24D* morphant (C), s*ec24D* + *pkd2* morphant (D), BFA-treated wildtype embryo (E) and BFA-treated *pkd2* morphant (F) showing no apoptosis at 48 hpf.



Supplementary figure 7: BFA-treated embryos have shortened body length. BFA-treated wildtype embryos have reduced body length (B) compared to non-treated embryos (A) at 48 hpf.



Supplementary figure 8: Vesicle trafficking inhibitor Cl-976 rescues axis curvature in *pkd2* morphants. 24 hpf *pkd2* morphant embryos were treated with the indicated concentrations of Cl-976 and scored for curvature at 30 hpf. 5 µM and 10 µM Cl-976 caused a significant reduction in embryos with moderate curvature compared to control embryos (* : p=0.0268; ** : p=0.0073)



Supplementary figure 9. Western blot quantification of phospho-S6 levels in *pkd2* morphants. phospho-S6 was compared to total S6 protein in control and *pkd2* morphant embryos at 24 hpf (left panel) and 48 hpf (right panel). Bar graphs show two independent experiments performed in triplicate for each condition.