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In our analysis of the zygotic DaPKC loss-of-function phenotype, we reported that embryos homozygous for the DaPKCk06403 allele die during early embryonic stages and fail to properly establish apical–basal polarity in embryonic epithelia and neuroblasts. Subsequent experiments in our lab showed that this early zygotic phenotype was only observed in the genetic background of the original fly stock that we used for our analysis. In a different genetic background, animals homozygous for the DaPKCk06403 allele survive until larval stages due to the maternal supply of DaPKC activity (Kim, S., and A. Wodarz, unpublished data; see Rolls et al., 2003). Upon removal of the maternal and zygotic activity of DaPKC in germ line clones of the DaPKCk06403 allele and of four new EMS alleles of DaPKC, we observed a fully penetrant loss of apical–basal polarity in embryonic epithelia and neuroblasts (Kim, S., B. Moussian, S. Luschnig, and A. Wodarz, unpublished data). We conclude that genetic background effects contributed to the early onset of the zygotic DaPKC loss-of-function phenotype that we originally reported. Nonetheless, our new data clearly show that DaPKC is indeed required for the control of apical–basal polarity in embryonic epithelia and neuroblasts.

We apologize to the readers for any confusion that may have been caused by this inaccuracy in our previous report.

Reference: Rolls, M.M., R. Albertson, H.P. Shih, C.Y. Lee, and C.Q. Doe. 2003. *Drosophila* aPKC regulates cell polarity and cell proliferation in neuroblasts and epithelia. *J. Cell Biol.* 163:1089–1098.