

LKB1 and AMPK maintain epithelial cell polarity under energetic stress

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The editors of *The Journal of Cell Biology* have been notified by Dr. Daniel St Johnston and Dr. Jay E. Brenman that they and the other authors of the paper referenced above retract the paper. As a result of this retraction, no data in this paper should be cited in the scientific literature.

The authors state:

Although the identification of lethal point mutations in the *Drosophila ampkα* gene and the originally identified neuronal phenotype (Fig. 1) remain valid, in follow-up experiments we have discovered that the defects in polarity marker localization, in actin distribution, and in epithelial integrity reported for *lkb1* and *ampk* mutant clones in the ovary follicular epithelium under starvation conditions (Fig. 2, A–C, and Fig. 4) result from an artefact. In short, follicular cell clones that should contain GFP can become damaged and create “false clones” of GFP-negative cells with the above-described defects. Bona fide *lkb1* and *ampk* mutant clones marked using a different technique do not show these phenotypes. As a result, our previous conclusion that LKB1 and AMPK function are required to maintain epithelial polarity and epithelial integrity under conditions of energetic stress is incorrect. Our conclusion that expression of *AMPKα-T184D* can rescue these defects in *lkb1* mutant clones (Fig. 5) therefore also is incorrect. We have further described this damage-induced artefact in a separate publication (Haack et al., 2013).

Not affected by this artefact are the following results reported in Mirouse et al. (2007): the identification and neuronal characterization of *ampkα* mutant alleles (Fig. 1); the absence of polarity defects in *tend* mutant clones (Fig. 2 D) and in *ampkα*³ mutant clones from flies grown on a glucose-only diet (Fig. 2 E); the localization of Cherry-AMPKα and GFP-LKB1 in wild-type follicle cells (Fig. 3 A); and the characterization of the anti-PhosphoT184-AMPKα antibody and its use to localize activated AMPKα in wild-type cells (Fig. 3 B). The *ampkα* transgenic *Drosophila* animals and *ampkα* alleles generated in the study remain suitable for use, and their description is now presented in a separate publication (Swick et al, 2013).

We apologize for any inconveniences that these erroneous conclusions may have caused.

References

- Haack, T., D.T. Bergstralh, and D. St Johnston. 2013. Damage to the *Drosophila* follicle cell epithelium produces “false clones” with apparent polarity phenotypes. *Biol. Open*. doi: 10.1242/bio.20134671.
- Swick, L.L., N. Kazgan, R. Uche Onyenwoke, and J.E. Brenman. 2013. Isolation of AMP-activated protein kinase (AMPK) alleles required for neuronal maintenance in *Drosophila melanogaster*. *Biol. Open*. doi: 10.1242/bio.20136775.