## Correction: Synaptopodin couples epithelial contractility to a-actinin-4-dependent junction maturation

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*The Journal of Cell Biology* regrets that Figs. 5, 6, 7, and 8 were originally published with mistakes. Specifically, the arrowheads in Figs. 5 (E and H), 6 (B–D), 7 K, and 8 A appeared at a larger size that obscured the data they pointed to. This was not the fault of the authors.

The authors regret that some of the references to Fig. 1 in the text refered to the incorrect panels. The corrected section appears below.

 $\alpha$ -Actinin-4 is required for vinculin recruitment and junction maturation

During normal epithelial maturation, actin and  $\alpha$ -actinin-4 accumulated at the cell junction over a period of several days (Fig. 1 A). By the second day post-confluence (2 dpc), most canonical junctional components, E-cadherin,  $\alpha$ -catenin, and  $\beta$ -catenin, p120-catenin, and ZO-1, were already present (Fig. 1, A and B). However, vinculin has not been targeted at this early stage of junction development (Fig. 1 B). By 5 dpc,  $\alpha$ -actinin-4 and vinculin became localized to the cell junction (Fig. 1 B). During this maturation period, the permeability barrier of the epithelial cell monolayer gradually formed (Fig. 1 C). Knockdown of  $\alpha$ -actinin-4 prevented this maturation process and compromised the development of the barrier function (Fig. 1 C), indicating that  $\alpha$ -actinin-4 recruitment is part of a normal maturation process during junction development. We have previously built a pressure chamber device that can deliver hydrostatic pressure to the intercellular junction to study the strength of cell–cell adhesion in an epithelial cell monolayer (Tang and Brieher, 2013). Using this setup, we show that intercellular stress induces  $\alpha$ -actinin-4 knockdown cells to break away from each other (Fig. 1 D) and eventually detach from the monolayer (Fig. 1 E), suggesting that cell–cell adhesion has been compromised.

Knockdown of  $\alpha$ -actinin-4 abolished junctional accumulation of vinculin (Fig. 1 F) without changing the cellular levels of vinculin (Fig. 1 G). Thus,  $\alpha$ -actinin-4 is incorporated into the maturing junctional complex before vinculin targeting and behaves as an upstream regulator of vinculin at the cell junction during junction maturation.

All of these corrections have been made to the HTML and PDF versions of this paper.