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Ptf1a control of Dll1 reveals an alternative to the lateral inhibition mechanism Jonas Ahnfelt-Rønne, Mette C. Jørgensen, Rasmus Klinck, Jan N. Jensen, Ernst-Martin Füchtbauer, Tye Deering, Raymond J. MacDonald, Chris V. E. Wright, Ole D. Madsen and Palle Serup

There was an error published in *Development* **139**, 33-45.

On p. 36, Fig. 1 was incorrectly cited several times in place of Fig. 2. The correct paragraph appears below.

The authors apologise to readers for this mistake.

To determine if endodermal Notch activation and *Hes1* expression depends on Dll1 activity, we analyzed NICD expression in wild-type and $Dll1^{lacZ/lacZ}$ embryos and EGFP expression in crosses of $Tg(Hes1-EGFP)^{lHri}$ and $Dll1^{lacZ/lacZ}$ embryos compared with controls (Fig. 2A,B), but appeared to recover, approaching wild-type levels at E11.5 (Fig. 2C,D). Hes1-EGFP expression was normal in E8.25 $Dll1^{lacZ/lacZ}$ embryos (Fig. 2E,I) but had partly disappeared from the dorsal pancreas endoderm at E9.5 (Fig. 2F,J) and was almost lost at E10.5 (Fig. 2G,K). Remarkably, and coinciding with the reappearance of NICD, Hes1-EGFP expression was restored in E11.5 $Dll1^{lacZ/lacZ}$ embryos (Fig. 2H,L).