

with the exception that CHIP assays were performed on cells harvested 72 h following a single transfection with dsRNA. *dRing* dsRNA including exonic sequences extending from 167 to 1,154 base pairs (bp) downstream of the ATG was synthesized by bi-directional transcription of RT-PCR products containing T7 promoter sequences at both ends. Isolation of wing imaginal discs and ChIP assays were performed as previously described²².

Information for purification and identification of histone H2A ubiquitin ligase complex, for generation and characterization of Ring2 knock-down cell lines, as well as for the specificity of the uH2A antibody, is available in Supplementary Methods and Supplementary Data.

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corrigenda

The lipid phosphatase SHIP2 controls insulin sensitivity

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In this Letter, we investigated the production and the phenotypic characterization of a SHIP2 (SH2 domain containing inositol phosphate 5-phosphatase type 2, or *Inpp11*) knockout mice. Total or partial loss of SHIP2 enzyme in these mice resulted in an increased insulin sensitivity. From these experiments, we concluded that SHIP2 is a potent negative regulator of insulin signalling and insulin sensitivity *in vivo*. However, we have recently realized that the 7.3-kilobase genomic DNA fragment deleted in these mice includes, in addition to exons 19–29 of the *SHIP2* gene, the third (and last) exon of the *Phox2a* gene. The deletion of this exon results in the absence of the 124 carboxy-terminal amino acids from a total of 280, including part of the homeodomain, and should give rise to a completely non-functional Phox2a protein if expressed. As a consequence, the mice we described have both *SHIP2* and *Phox2a* genes inactivated. It is currently unknown whether the increased insulin sensitivity we observed in our mice results from the inactivation of the *SHIP2* gene alone, of the *Phox2a* gene alone, or of both genes. □

Induction of DNA methylation and gene silencing by short interfering RNAs in human cells

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In the Methods section of this Letter, the published primer sequences used to amplify the E-cadherin and *erbB2* promoters for bisulphite sequencing were incorrect. We used two primer sets, one for unconverted DNAs and the other for converted DNAs. Primers for unconverted DNAs were: for the E-cadherin promoter, the forward primer was 5'-TCTAGAAAAATTTTAAAAA-3' and the reverse primer was 5'-CAGCGCCGAGAGGCTGCGCT-3'; for the *erbB2* promoter, the forward primer was 5'-CCTGGAAGCCA-CAAGGTAAAC-3' and reverse primer was 5'-TTTCTCCGG TCCCAATGGAGG-3'. Primers for converted DNAs were: for the E-cadherin promoter, the forward primer was 5'-TTTA-GAAAAATTTTAAAAA-3' and the reverse primer was 5'-CAA-CACCAAAAACTACAAC-3'; for the *erbB2* promoter, the forward primer was 5'-TTTGAAGTTATAAGGTAAAT-3' and the reverse primer was 5'-TTTCTCCAATCCCAATAAAAA-3'. □