RETRACTION

Ramalingam, T. S., West, A. P., Lebron, J. A., Nangiana, J. S., Hogan, T. H., Enns, C. A. & Bjorkman, P. J. Binding to the transferrin receptor is required for endocytosis of HFE and regulation of iron homeostasis. *Nature Cell Biol.* **2,** 953–957 (2000).

In the course of repeating experiments described in the above paper, we have discovered that some of the confocal microscopy images presented in Figs 1 and 2 were inappropriately processed. A major point of the paper is that wild-type HFE expressed in HuTu-80 cells colocalizes with transferrin receptor (TfR) on the basolateral membrane and in intracellular transferrin (Tf)-positive endosomes, whereas a mutant form of HFE (HFE^{W81A}) that binds TfR with reduced affinity is localized mainly at the basolateral membrane and not in Tf-positive endosomes. In correctly processed images, we find HFE^{W81A} both at the basolateral membrane and in Tf-positive vesicles. Although the amount of HFE^{W81A} at the basolateral membrane versus HFE^{W81A} in intracellular vesicles is increased when compared with wild-type HFE (N.E. Tiangco and P.J.B., unpublished observations), the differences in HFE localization between cells expressing wild-type HFE and HFE^{W81A} are subtle. The paper also presents the results of western blots to measure ferritin levels in untransfected cells and cells expressing wild-type HFE, HFE^{W81A}, the hemochromatosis mutant HFE^{C260Y}, and wild-type and W81A forms of HFE to which the endosomal sorting sequence from the low-density lipoprotein receptor (LDLR) has been attached. Our current results are in agreement with the findings of the paper: that is, that ferritin levels are lower in HuTu-80 cells expressing wild-type HFE and wild-type HFE–LDLR than in untransfected HuTu-80 cells and in cells expressing HFE^{W81A}, HFE^{W81A}–LDLR or HFE^{C260Y} (A.-S. Zhang and C.A.E., unpublished observations). However, the differences between the present confocal images and the ones published with the paper require us to revise the conclusion that TfR binding by HFE is required for endocytosis of HFE and HFE-mediated regulation of ferritin levels, and we are therefore retracting the paper. We deeply regret any inconvenience this publication has caused for others.

The first author of this paper (T.S.R.) is not a cosignee of this retraction.

ERRATA

In Saucedo *et al.* (*Nature Cell Biol.* **5**, 566–571 (2003)), the first two sentences of the abstract should read:

Insulin signalling is a potent **stimulator** of cell growth and has been proposed to function, at least in part, through the conserved protein kinase TOR (target of rapamycin). Recent studies suggest that the tuberous sclerosis complex Tsc1–Tsc2 may couple insulin signalling to Tor activity.

rather than:

Insulin signalling is a potent **inhibitor** of cell growth and has been proposed to function, at least in part, through the conserved protein kinase TOR (target of rapamycin). Recent studies suggest a that the tuberous sclerosis complex Tsc1–Tsc2 may couple insulin signalling to Tor activity. This has also been corrected online.

In Guglielmo *et al.* (*Nature Cell Biol.* **5,** 410–421 (2003)), on p414, second column, line 9, the text should read:

Autoradiography (Fig. 1g) and quantitation (Fig. 1h) demonstrated that receptors were in the raft (55%) and non-raft fraction, which contained caveolin-1 or EEA1, respectively.

rather than:

Autoradiography (Fig. 1g) and quantitation (Fig. 1g) demonstrated that receptors were in the raft (55%) and non-raft fraction, which contained caveolin-1 or EEA1, respectively.

This has also been corrected online.

CORRIGENDUM

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hDia2C       1 MEQPGAAASGAGGGSEEPGGGRSNKRSAGNRAANEEETKNKPKL-----------RDRITSFRKSTVKKEKPLIQHPIDSQVAMSEFPAA
hDia2B       1 MEQPGAAASGAGGGSEEPGGGRSNKRSAGNRAANEEETKNKPKLNIQIKTLADDVRDRITSFRKSTVKKEKPLIQHPIDSQVAMSEFPAA
mDia2        1 MERHRARALGRDSKSSRRKGLQSAPPAGPYEPGEKRPKLHLN-----IRTLTDDMLDKFASIRIPGSKKERPPLPHLKTVSGISDSSSLS
mDia1        1 MEPSGGGLGPGRGTRDKKKGRSPDELPATGGDGGKHKKFLER-------------------FTSMRIKKEKEKP----NSAHRNSSASYG
consensus    1 **............... *............. .. ....     .... ...............***............... . ..

hDia2C      80 QPLYDERSLNLSEKEVLDLFEKMMEDMNLNEEKKAPLRNKDFTTKREMVVQYISATAKSIVGSKVTGGLKNSKHECTLSSQEYVHELRSG
hDia2B      91 QPLYDERSLNLSEKEVLDLFEKMMEDMNLNEEKKAPLRNKDFTTKREMVVQYISATAK-------SGGLKNSKHECTLSSQEYVHELRSG
mDia2       86 SETMENNPKALPESEVLKLFEKMMEDMNLNEDKKAPLREKDFGIKKEMVMQYINTASKTG----------SLRSSRQISPQEFLHELKMG
mDia1       68 DDPTAQSLQDISDEQVLVLFEQMLVDMNLNEEKQQPLREKDIVIKREMVSQYLHTSKAG-----------MNQKESSRSAMMYIQELRSG
consensus   91 ...............**.***.*..******.*..*** **.. *.***.**.. ....      .............*......**..*
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In Gasman *et al.* (*Nature Cell Biol.* **5**, 195–204 (2003)), the correct sequence alignment in Fig. 1a should be:

Consequently, on page 197, the text in the right column, line 2, should read "... deletion of 11 (45–55 of hDia2B exon 12C and 156) ...". Similarly, the text on line 16 should read "... amino acids 45–55 of hDia2-12C ...". These changes do not significantly alter the conclusions and conceptual message of the paper. This has also been corrected online.