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CORRECTION

Correction to: Hepcidin Mediates Transcriptional Changes in Ferroportin mRNA in Differentiated Neuronal-like PC12 Cells Subjected to Iron Challenge

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The original version of this article unfortunately contained mistakes on Figs. 1, 2, and 7 as some of the data were not visible. With this, the correct images are hereby published.

In Table 4 footnote, "Forty millimolar40 mM" should be changed two times to "Forty millimolar (40mM)". Also layout of Tables 2 - 4 were changed as per request of authors. Corrected Tables are presented herewith.

The online version of the original article can be found at https://doi.org/10.1007/s12035-018-1241-3

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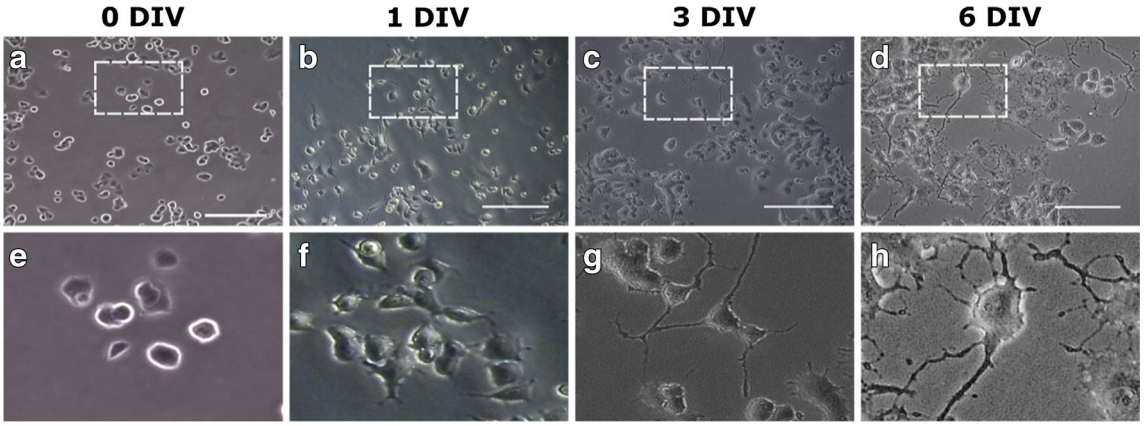

Fig. 1 **a**–**d** Growth properties of PC12 cells before (**a**) and after receiving 50 ng/mLNGF-β for 1, 3, and 6 daysDIV(**b**–**d**). Eighty to ninety percent of the cells display differentiated morphology after 6 DIV. **e**–**h** Areas marked with respective squares in **a**–**d** shown in higher magnification obtained by computed enlargement. The PC12 cells differentiate into cells with morphological changes corresponding to neurons with polygonal cell bodies and extended cellular processes with several branches sharing the morphology of mature neurites. Scale bar = 200 μm

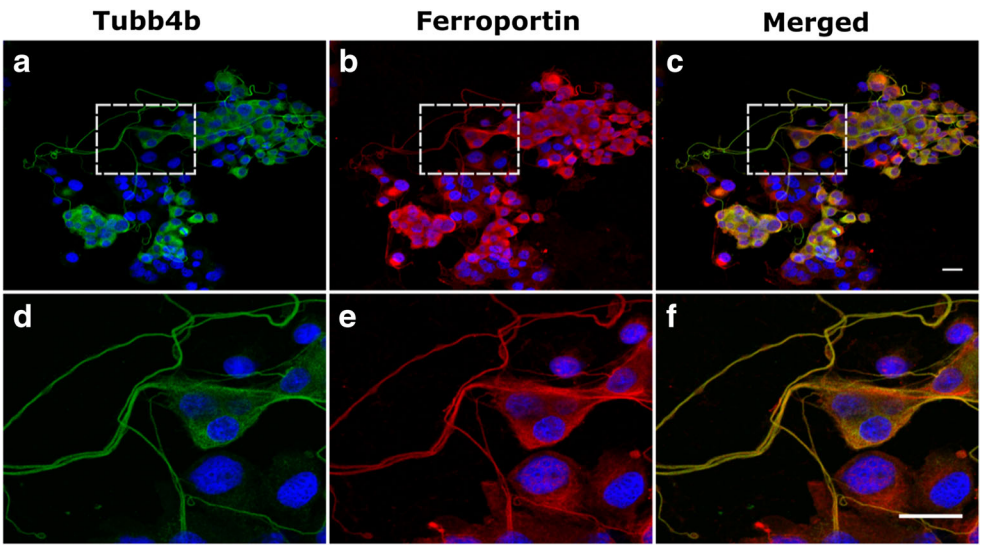

Fig. 2 **a**–**b** Confocal images of differentiated PC12 cells after 6 days of treatment with NGF-β revealing protein expression and cellular localization of Tubb4b (**a**, **d**) (green) and ferroportin (**b**, **e**) (red). Nuclei are counterstained with DAPI (blue). **d**–**e** Areas marked with respective squares in **a**–**c** shown in higher magnification obtained by computed enlargement. The extended processes of the differentiated PC12 cells are clearly seen in these high-power magnifications. c, f Merged photos. Scale bar = 20 μm

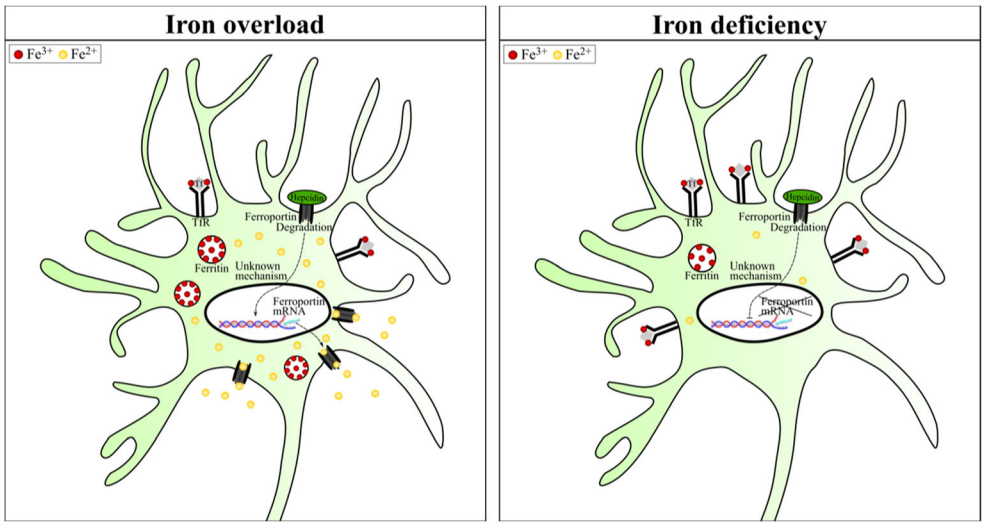

Fig. 7 Overview of our hypothesis regarding the action of hepcidin on *Fpn* mRNA in NGF-β differentiated PC12 cells depending on the iron status. (Left) In iron overload, a strong upregulation of *Fpn* mRNA is mediated by hepcidin. This could lead to increase translation of *Fpn* mRNA and hence more ferroportin protein (yellow dots) in spite hepcidin also provokes a degradation of the ferroportin when present in the cellular membrane. A raised cytosolic appearance of ferroportin may nonetheless aid capture of excess iron and prevent ROS-mediated damage caused by unbound intracellular iron. (Right) In cellular iron deficiency, hepcidin mediates a decrease in *Fpn* mRNA expression, which lowers the translational and presence of ferroportin protein (yellow dots) in the cellular membrane. In turn, this increases the survival of the cell by retaining iron intracellularly to facilitate its availability

Table 2 Primers used for RT-qPCR

Target	Forward primer	Reverse primer
Fth	GACCACCGCGTCTCCCTCGC	CAGGGCCACATCATCCCGGTC
Ftl	TGACGTGGCTTTGGAAGGCG	GATGGCTTCTGCACATCCTGG
Hdac1	GCTGAGGAGATGACCAAG	GTGGACAACTGACAGAAC
Phf8	CCAGAAAGCAAAGCTCAA	GCACTGTCTACCTTCTTC
Tet1	GAGTCTTCACCATGACAC	GGACATGAATTCTTAGAACTATC

Table 3 The expression of *Fpn* mRNA after treatment with various concentrations of FAC in combination with a fixed concentration of hepcidin. Values below 1 indicate downregulation of *Fpn* mRNA expression, and values above 1 the reverse.

Treatment group	Ratio (treatment/control)	Effect on *Fpn* mRNA	Significance
FAC 10 mM	0.889	(↓)	n.s
FAC 20 mM	1.49	(↑)	n.s
FAC 30 mM	5.97	(↑)	n.s
FAC 6 mM + 1.0 μM hepcidin	14.122	(↑)	n.s
FAC 20 mM + 1.0 μM hepcidin	95.14	↑	***
FAC 20 mM + 1.0 μM hepcidin/FAC 20 mM	93.92	↑	***

The expression of Fpn after treatment with FAC is normalized to that of the control group receiving no treatment, whereas the expression of Fpn after treatment with FAC in combination with hepcidin is normalized to the group receiving 1.0 μM hepcidin. Effects presented in brackets display tendency without significance. Combining 20 mM FAC and 1.0 μM hepcidin upregulates the expression of Fpn, approximately times 90. A comparison between the treatments with 20 mM FAC and 20 mM FAC in combination with 1.0 μM hepcidin, measured as the ratio of expression, demonstrates a significant increase mediated by hepcidin to the two situations with identical concentrations of FAC. Significance: ***p = 0.0001—0.001, n.s. not significant.

Table 4 The expression of *Fpn* mRNA after treatment with 40 mM DFO in combination with 2.3 μM hepcidin.

Treatment group	Ratio (treatment/control)	Effect on *Fpn* mRNA	Significance
DFO 40 mM	8.81	↑	*
DFO 40 mM + 2.3 μM hepcidin	0.42	↓	n.s.
DFO 40 mM/DFO 40 mM +2.3 μM hepcidin	20.63	↑	**

Values below 1 indicate a downregulation of Fpn mRNA expression, and values above 1 the reverse. The expression of Fpn after treatment with DFO is normalized to that of the control group receiving no treatment, whereas the expression of Fpn after treatment with DFO in combination with hepcidin is normalized to the group receiving 2.3 μM hepcidin. Forty millimolar (40mM) DFO upregulates Fpn significantly. Forty millimolar (40mM) DFO combined with 2.3 μM hepcidin results in downregulation of Fpn mRNA. A comparison between the treatments with (40mM) DFO and (40mM) DFO + 2.3 μM hepcidin, measured as the ratio of expression, reveals strong upregulation of Fpn mRNA in the single treatment group without hepcidin with an approximately 20 times higher expression. Significance: *p = 0.01—0.05, ** p = 0.001—0.01, n.s. not significant.