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SPECIALTY SECTION This article was submitted to Signaling, a section of the journal Frontiers in Cell and Developmental Biology

RECEIVED 11 December 2022 ACCEPTED 22 December 2022 PUBLISHED 06 January 2023

CITATION

Ren Y, Liu W, Zhang J, Bi J, Fan M, Lv Y, Wu Z, Zhang Y and Wu R (2023), Corrigendum: MFG-E8 maintains cellular homeostasis by suppressing endoplasmic reticulum stress in pancreatic exocrine acinar cells. *Front. Cell Dev. Biol.* 10:1121052. doi: 10.3389/fcell.2022.1121052

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Corrigendum: MFG-E8 maintains cellular homeostasis by suppressing endoplasmic reticulum stress in pancreatic exocrine acinar cells

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KEYWORDS

pancreatic exocrine acinar cells, endoplasmic reticulum stress, acute pancreatitis, MFG-E8, $\alpha\nu\beta3/5$ integrins, FAK-STAT3 pathway

A Corrigendum on

MFG-E8 maintains cellular homeostasis by suppressing endoplasmic reticulum stress in pancreatic exocrine acinar cells

by Ren Y, Liu W, Zhang J, Bi J, Fan M, Lv Y, Wu Z, Zhang Y and Wu R (2022). Front. Cell Dev. Biol. 9: 803876. doi: 10.3389/fcell.2021.803876

In the original article, there was a mistake in Figures 2, 3 as published. The TEM results in the Sham group in Figure 2A inadvertently used the Sham group of mfge8-KO mice. In Figure 3A, the image of MPO with Vehicle treatment was misused from pancreas in CD11b group. The corrected Figures 2, 3 and its caption appear below:

The authors apologize for this error and state that this does not change the scientific conclusions of the article in any way. The original article has been updated.



FIGURE 2

Exogenous MFG-E8 alleviates pancreatic ER stress *in vivo*. In mice, arginine-AP stress was induced by 2 h intraperitoneal injections of 4.0 g/kg L-arginine. At 2 h after the last injection of L-arginine, normal saline (vehicle) or 20 μ g/kg MFG-E8 were administered through intraperitoneal injection. The animals were sacrificed at 69 h after MFG-E8 treatment (i.e., 72 h after the first injection of L-arginine). Blood and tissue samples were collected. **(A)** Ultrastructural alterations in the pancreas (Transmission electron microscopy); **(B,C)** Representative photos of GRP78 staining and quantitative of GRP78 staining; **(D)** Western blot analysis of the expression of GRP78, phospho-IRE1 α and IRE1 α in the pancreas; **(E)** Western blot analysis of the expression of phospho-PERK, PERK, phospho-elF2 α and elF2 α in the pancreas; **(F)** Western blot analysis of the expression of CHOP, caspase-9 and cleaved caspase-9 in the pancreas. *n* = 4–6/group, error bars indicate the SEM; * *p* < .05 *versus* Sham group; #*p* < .05 *versus* Vehicle group. MFG-E8, milk fat globule EGF factor 8; AP, acute pancreatitis; GRP78, glucose-regulated protein 78; elF2 α , eukaryotic initiation factor 2 α ; ATF-6, Activating Transcription Factor 6; CHOP, C/EBP homologous protein; Nucl, nucleus; Cyto, cytoplasm; H-3, histone-3; PERK, PKR-like endoplasmic reticulum kinase.



FIGURE 3

MFG-E8 alleviates the inflammatory response in experimental-AP through NF- κ B signaling pathway. In mice, arginine-AP stress was induced by 2 h intraperitoneal injections of 4.0 g/kg L-arginine. At 2 h after the last injection of L-arginine, normal saline (vehicle) or 20 µg/kg MFG-E8 were administered through intraperitoneal injection. The animals were sacrificed at 69 h after MFG-E8 treatment (i.e., 72 h after the first injection of L-arginine). Blood and tissue samples were collected. (A) Representative photos of F4/80, CD11b, Gr1 and MPO staining; (B) Quantitative of F4/80, CD11b, Gr1 and MPO staining; (C) Serum TNF- α , IL-6 and HMGB-1 levels; (D,E) Western blot analysis of the expression of phospho-IkB α , IkB α , nucl-P65, cyto-P65, nucl-H3 and cyto- β -actin in the pancreas. *n* = 4–6/group, error bars indicate the SEM; * *p* < .05 versus Sham group; #*p* < .05 versus Vehicle group. MFG-E8, milk fat globule EGF factor 8; AP, acute pancreatitis; MPO, myeloperoxidase; HMGB-1, High mobility group box 1; Nucl, nucleus; Cyto, cytoplasm; H-3, histone-3; IkB α , inhibitor of NF- κ B p65, Nuclear Factor Kappa-B p65.

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