



OPEN ACCESS

EDITED AND REVIEWED BY
Dieter Steinhilber,
Goethe University Frankfurt, Germany

*CORRESPONDENCE
Fengjing Guo,
✉ guofjdoc@163.com

[†]These authors have contributed equally
to this work

RECEIVED 04 April 2023
ACCEPTED 20 April 2023
PUBLISHED 12 May 2023

CITATION

Guo Z, Lin J, Sun K, Guo J, Yao X, Wang G,
Hou L, Xu J, Guo J and Guo F (2023),
Corrigendum: Deferoxamine alleviates
osteoarthritis by inhibiting chondrocyte
ferroptosis and activating
the Nrf2 pathway.
Front. Pharmacol. 14:1199951.
doi: 10.3389/fphar.2023.1199951

COPYRIGHT

© 2023 Guo, Lin, Sun, Guo, Yao, Wang,
Hou, Xu, Guo and Guo. This is an open-
access article distributed under the terms
of the [Creative Commons Attribution
License \(CC BY\)](https://creativecommons.org/licenses/by/4.0/). The use, distribution or
reproduction in other forums is
permitted, provided the original author(s)
and the copyright owner(s) are credited
and that the original publication in this
journal is cited, in accordance with
accepted academic practice. No use,
distribution or reproduction is permitted
which does not comply with these terms.

Corrigendum: Deferoxamine alleviates osteoarthritis by inhibiting chondrocyte ferroptosis and activating the Nrf2 pathway

Zhou Guo^{1†}, Jiamin Lin^{1†}, Kai Sun¹, Jiayou Guo², Xudong Yao³,
Genchun Wang¹, Liangcai Hou¹, Jingting Xu¹, Jiachao Guo⁴ and
Fengjing Guo^{1*}

¹Department of Orthopedics, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China, ²Michigan State University's Broad College of Business, East Lansing, MI, United States, ³Department of Orthopedic Surgery, The Second Affiliated Hospital of Chongqing Medical University, Chongqing, China, ⁴Department of Pediatric Surgery, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

KEYWORDS

osteoarthritis, chondrocytes, ferroptosis, deferoxamine, Nrf2

A Corrigendum on

Deferoxamine alleviates osteoarthritis by inhibiting chondrocyte ferroptosis and activating the Nrf2 pathway

by Guo Z, Lin J, Sun K, Guo J, Yao X, Wang G, Hou L, Xu J, Guo J and Guo F (2022). *Front. Pharmacol.* 13:791376. doi: 10.3389/fphar.2022.791376

In the published article, there was an error in (Figure 3H) as published. (Due to the error of data filing, the fluorescence images of the Erastin group in Figure 3H was inserted incorrectly). The corrected (Figure 3H) and its caption (Erastin initiated inflammation responses and ECM degradation in chondrocytes that could be alleviated by Ferrostatin-1) appear below.

In the published article, there was an error in (Figure 5F) as published. (Due to the error of data filing, the fluorescence images of the Control group in Figure 5F was inserted incorrectly). The corrected (Figure 5F) and its caption (DFO alleviated chondrocytes ferroptosis and OA progress induced by erastin) appear below.

In the published article, there was an error. (There is a clerical error in the Materials and Methods section).

A correction has been made to (Materials and Methods), (Animal experiment). This sentence previously stated:

“(the sham group, DMM group, DMM + DFO (10 mg/kg) group, DMM + DFO (100 mg/kg) group, erastin group, erastin (15 mg/kg) + DFO (10 mg/kg) group, and erastin (15 mg/kg) + DFO (100 mg/kg) group.)”

The corrected sentence appears below:

“(the sham group, DMM group, DMM + DFO (10 mg/kg) group, DMM + DFO (100 mg/kg) group, erastin group, erastin (5 mg/kg) + DFO (10 mg/kg) group, and erastin (5 mg/kg) + DFO (100 mg/kg) group.)”

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

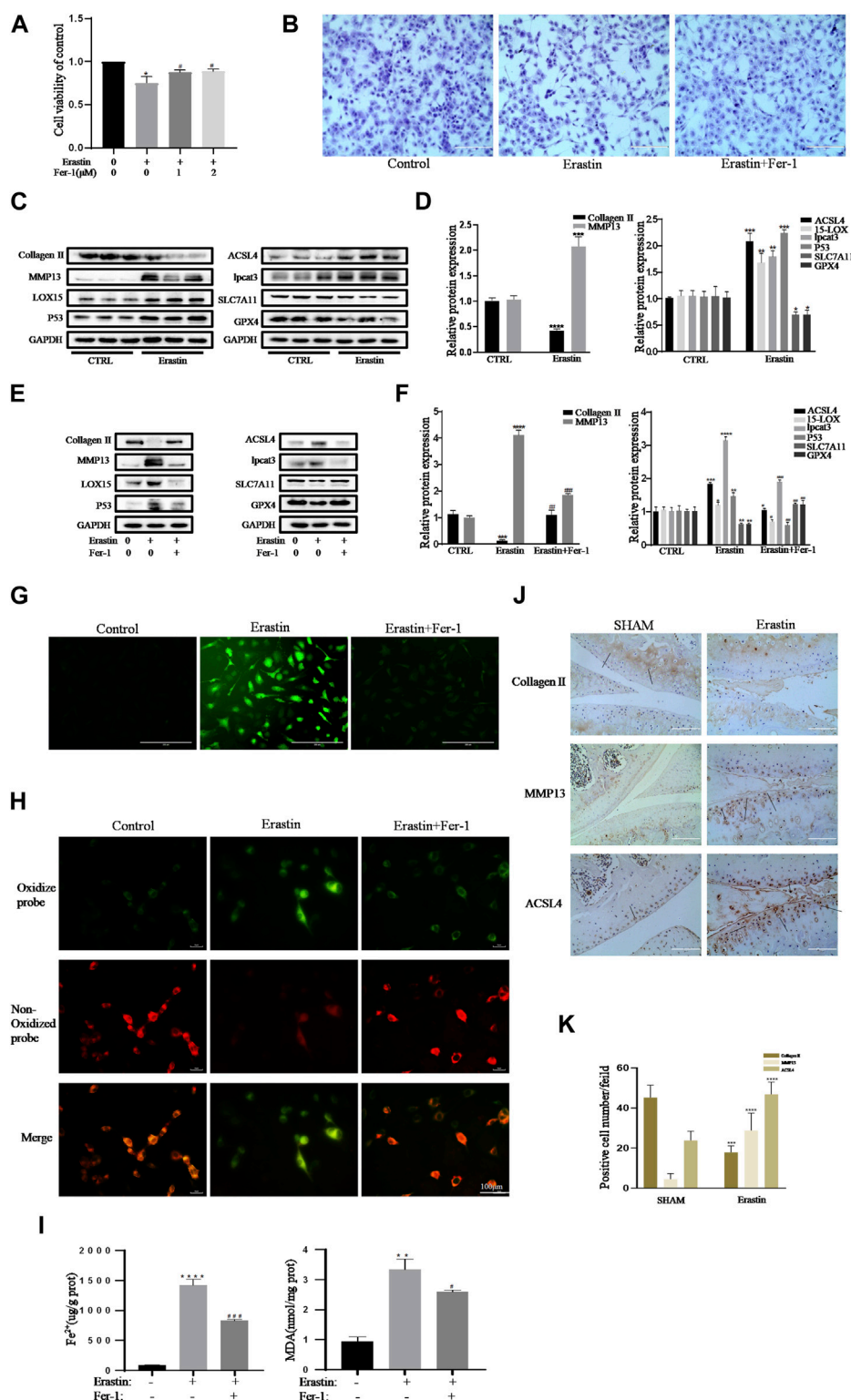


FIGURE 3

Erastin initiated inflammation responses and ECM degradation in chondrocytes that could be alleviated by Ferrostatin-1. **(A,B)** Cell viability determined by CCK-8 assay and toluidine blue staining. **(C,D)** The protein expression levels of collagen II, MMP13, ACSL4, LOX15, lpcat3, P53, and SLC7A11 GPX4 when treated by erastin (5 μM) were detected by Western blot, and band density ratios of collagen II, MMP13, ACSL4, LOX15, lpcat3, P53, and SLC7A11 GPX4 to GAPDH in the Western blots were quantified by densitometry ($n = 3$). **(E,F)** The protein expression levels of collagen II, MMP13, ACSL4, LOX15, lpcat3, P53, and SLC7A11 GPX4 when treated by Erastin (5 μM) with fer-1 (1 μM) or equal volume of DMSO were detected by Western blot, and band density ratios of collagen II, MMP13, ACSL4, LOX15, lpcat3, P53, and SLC7A11 GPX4 to GAPDH in the Western blots were quantified by densitometry ($n = 3$). **(G)** Intracellular ROS level detected by DCFH-DA fluorescent probe (scale bar: 200 μm). **(H)** Intracellular lipid-ROS level detected by C11 BODIPY fluorescent probe (scale bar: 100 μm). Red, reduced form of C11-BODIPY; green, oxidized form of C11-BODIPY. **(I)** The intracellular level of

(Continued)

FIGURE 3 (Continued)

MDA and Fe²⁺ was determined using the MDA assay kit and iron assay kit (n = 3). **(J)** The collagen II, MMP13, and ACSL4 expression levels in the cartilage samples were measured using immunohistochemistry staining. Dotted arrows indicate positive cells for MMP13 and ACSL4 and positive staining of collagen II (scale bar: 100 μm). **(K)** Quantification of MMP13- and ACSL4-positive cells and collagen II-positive staining in vivo. *p < 0.05 versus control or the sham group, **p < 0.01 versus control or the sham group, ***p < 0.001 versus control or the sham group, ****p < 0.0001 versus control or the sham group, #p < 0.05 versus IL-1β-treated group, ##p < 0.01 versus IL-1β-treated group, and ###p < 0.001 versus IL-1β-treated group. Error bars represent SD.

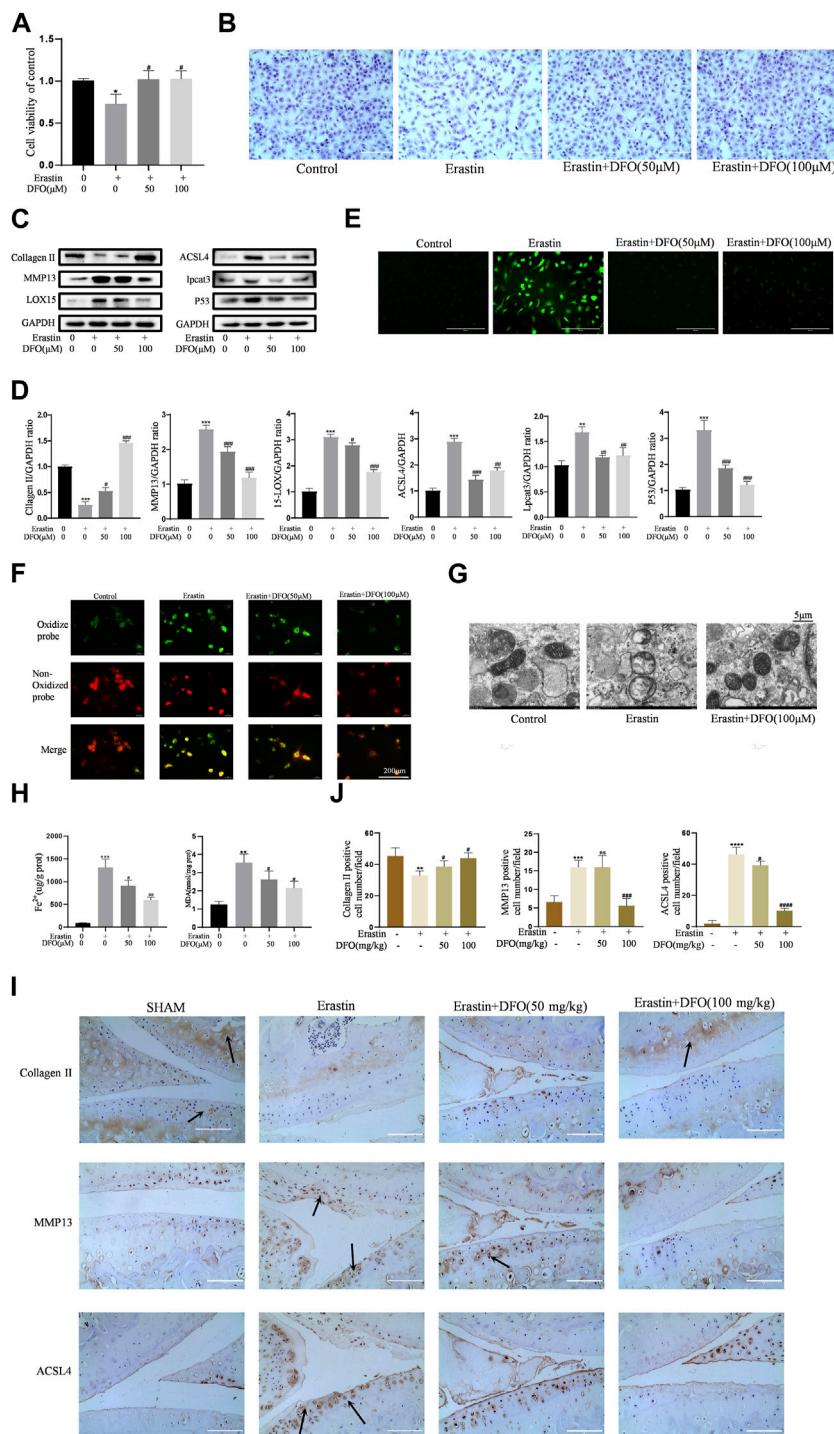


FIGURE 5

DFO alleviated chondrocytes ferroptosis and OA progress induced by erastin. **(A,B)** Cell viability determined by CCK-8 assay and toluidine blue staining. **(C,D)** The protein expression levels of collagen II, MMP13, ACSL4, LOX15, lpcat3, and P53 when treated by Erastin (5 μM) with 50 and 100 μM DFO (Continued)

FIGURE 5 (Continued)

or equal volume of DMSO were detected by Western blot, and band density ratios of collagen II, MMP13, ACSL4, LOX15, lpcat3, and P53 to GAPDH in the Western blots were quantified by densitometry ($n = 3$). **(E)** Intracellular ROS level detected by DCFH-DA fluorescent probe (scale bar: 200 μm). **(F)** Intracellular lipid-ROS level detected by C11 BODIPY fluorescent probe (scale bar: 200 μm). Red, reduced form of C11-BODIPY; green, oxidized form of C11-BODIPY. **(G)** The ultrastructure of mitochondria observed via transmission electron microscopy (scale bar: 5 μm). **(H)** The intracellular level of MDA and Fe^{2+} was determined using the MDA assay kit and iron assay kit ($n = 3$). **(I)** The collagen II, MMP13, and ACSL4 expression levels in the cartilage samples were measured using immunohistochemistry staining. Dotted arrows indicate positive cells for MMP13 and ACSL4 and positive staining of collagen II (scale bar: 100 μm). **(J)** Quantification of MMP13- and ACSL4-positive cells and collagen II-positive staining in vivo. $*p < 0.05$ versus control or the sham group, $**p < 0.01$ versus control or the sham group, $***p < 0.001$ versus control or the sham group, $****p < 0.0001$ versus control or the sham group, $\#p < 0.05$ versus IL-1 β -treated group or the DMM group, $\##p < 0.01$ versus IL-1 β -treated group or the DMM group, $\###p < 0.001$ versus IL-1 β -treated group or the DMM group, and $\####p < 0.0001$ versus IL-1 β -treated group or the DMM group. Error bars represent SD.

Publisher's note

All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated

organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher.