Biochemical Society Transactions (2020) **48** 2915
https://doi.org/10.1042/BST-2020-0523C_COR

PORTLAND PRESS

Correction: Emerging aspects in the regulation of ferroptosis

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Biochem. Soc. Trans. (2020) 48 (5): 2253–2259 DOI: https://doi.org/10.1042/BST20200523

The authors of this paper would like to make the following correction to the citations within the text:

Reference 48 was a duplication of reference 12 and should have instead cited Bersuker, K., Hendricks, J.M., Li, Z. et al. (2019) The CoQ oxidoreductase FSP1 acts parallel to GPX4 to inhibit ferroptosis *Nature* **575**, 688–692. DOI: 10.1038/s41586-019-1705-2

In the section entitled "The FSP1 ubiquinone system"

> The FSP1-ubiquinone axis was then characterized as a GSH/GPX4 independent ferroptosis-suppressing pathway that exemplifies an elegant system for the enzymatic regeneration of endogenous antioxidants [47].

Should instead cite the following:

> The FSP1-ubiquinone axis was then characterized as a GSH/GPX4 independent ferroptosis-suppressing pathway that exemplifies an elegant system for the enzymatic regeneration of endogenous antioxidants [47, 48].

In the section entitled "The tetrahydrobiopterin-DHFR system"

> More recently, another novel endogenous antioxidant system has been identified. Using CRISPR activation screens, a study identified tetrahydrobiopterin (BH4) as an essential metabolite supporting the proliferation of cancer cell lines challenged with the GPX4 inhibitor RSL3 [48].

Should have cited

> More recently, another novel endogenous antioxidant system has been identified. Using CRISPR activation screens, a study identified tetrahydrobiopterin (BH4) as an essential metabolite supporting the proliferation of cancer cell lines challenged with the GPX4 inhibitor RSL3 [11, 12].

At the end of the article, the final sentence

> Hence, these studies demonstrate that DHFR should be regarded as an important regulator of ferroptosis, while its inhibitor methotrexate, a common chemotherapeutic agent, could be a promising therapeutic option for ferroptotic anti-cancer therapies [48]

Should have read

> Hence, these studies demonstrate that DHFR should be regarded as an important regulator of ferroptosis, while its inhibitor methotrexate, a common chemotherapeutic agent, could be a promising therapeutic option for ferroptotic anti-cancer therapies [12].

Correction published:
18 December 2020