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Corrigendum: Receptor of advanced glycation end products deficiency attenuates cisplatin-induced acute nephrotoxicity by inhibiting apoptosis, inflammation and restoring fatty acid oxidation

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A Corrigendum on

[Receptor of advanced glycation end products deficiency attenuates cisplatin-induced acute nephrotoxicity by inhibiting apoptosis, inflammation and restoring fatty acid oxidation](#)

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In the published article, there was an error in affiliation 1. Instead of “Department of Endocrinology, Xiang’an Hospital of Xiamen University, Xiamen, China”, it should be “Department of Internal Medicine, Xiang’an Hospital of Xiamen University, School of Medicine, Xiamen University, Xiamen, China”.

In the published article, the reference **Elimam et al.** was not cited. The citation has now been inserted in **Discussion**, Paragraph 6, and now reads:

“Apart from being a source of energy, fatty acids are also engaged in the formation of mitochondrial membrane phospholipids. Calcium-independent Phospholipase A2 γ can repair damaged mitochondrial membrane phospholipids by hydrolyzing damaged acyl chains to make them re-esterify with fatty acids and thus maintain mitochondrial survival and function, including FAO (Elimam et al., 2013).”

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.

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Reference

Elimam, H., Papillon, J., Takano, T., and Cybulsky, A. V. (2013). Complement-mediated activation of calcium-independent phospholipase A₂: Role of protein

kinases and phosphorylation. *J. Biol. Chem.* 288 (6), 3871–3885. doi:10.1074/jbc.M112.396614