



# Corrigendum: FGF23 in Cardiovascular Disease: Innocent Bystander or Active Mediator?

Robert Stöhr<sup>1\*</sup>, Alexander Schuh<sup>1</sup>, Gunnar H. Heine<sup>2</sup> and Vincent Brandenburg<sup>1</sup>

<sup>1</sup> Department of Cardiology, University Hospital of the RWTH Aachen, Aachen, Germany, <sup>2</sup> Department of Nephrology, University Hospital Homburg-Saar, Homburg, Germany

Keywords: FGF23, cardiovascular diseases, heart failure, hypertrophy, left ventricular, myocardial infarction

# A corrigendum on

### FGF23 in Cardiovascular Disease: Innocent Bystander or Active Mediator?

by Stöhr, R., Schuh, A., Heine, G. H., and Brandenburg, V. (2018). Front. Endocrinol. 9:351. doi: 10. 3389/fendo.2018.00351

# **OPEN ACCESS**

#### Approved by:

Frontiers in Endocrinology Editorial Office, Frontiers Media SA, Switzerland

# \*Correspondence:

Robert Stöhr rstoehr@ukaachen.de

#### Specialty section:

This article was submitted to Molecular and Structural Endocrinology, a section of the journal Frontiers in Endocrinology

Received: 29 June 2018 Accepted: 05 July 2018 Published: 18 July 2018

#### Citation:

Stöhr R, Schuh A, Heine GH and Brandenburg V (2018) Corrigendum: FGF23 in Cardiovascular Disease: Innocent Bystander or Active Mediator? Front. Endocrinol. 9:422. doi: 10.3389/fendo.2018.00422 In the original article, there was an error. The last paragraph contained several mistakes carried on from a previous version.

A correction has been made to the section Cardiac FGF23 Research: The Next Level, paragraph 2 and should read:

"Prior to any therapeutic intervention with the aim to minimize potentially negative FGF23 effects upon cardiac structure and function, research needs to focus on and clarify relevant unsolved issues. Just to name a few, the community needs to prove how cardiac disease induces (rather than follows) FGF-23 secretion, to what degree cardiomyocytes may themselves produce FGF-23 in health and disease, whether such locally produced FGF-23 has a physiological role in (acute) myocardial damage; and whether or not (systemic) FGF23 excess itself directly drives the development of myocardial damage. Only when these questions have been answered, can we try to discuss whether and how to intervene on serum FGF-23 level."

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.

**Conflict of Interest Statement:** The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Copyright © 2018 Stöhr, Schuh, Heine and Brandenburg. This is an open-access article distributed under the terms of the Creative Commons Attribution License (CC BY). 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.