

Erythropoietin induces bone marrow and plasma fibroblast growth factor 23 during acute kidney injury

Por: [Toro, L](#) (Toro, Luis)^[1,2,3]; [Barrientos, V](#) (Barrientos, Victor)^[1,4]; [Leon, P](#) (Leon, Pablo)^[1]; [Rojas, M](#) (Rojas, Macarena)^[1]; [Gonzalez, M](#) (Gonzalez, Magdalena)^[1]; [Gonzalez-Ibanez, A](#) (Gonzalez-Ibanez, Alvaro)^[4,5,6]; [Illanes, S](#) (Illanes, Sebastian)^[7]; [Sugikawa, K](#) (Sugikawa, Keigo)^[8]; [Abarzu, N](#) (Abarzu, Nestor)^[1]; [Bascunan, C](#) (Bascunan, Cesar)^[1] [...Más](#)

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Abstract

It is accepted that osteoblasts/osteocytes are the major source for circulating fibroblast growth factor 23 (FGF23). However, erythropoietic cells of bone marrow also express FGF23. The modulation of FGF23 expression in bone marrow and potential contribution to circulating FGF23 has not been well studied. Moreover, recent studies show that plasma FGF23 may increase early during acute kidney injury (AKI). Erythropoietin, a kidney-derived hormone that targets erythropoietic cells, increases in AKI. Here we tested whether an acute increase of plasma erythropoietin induces FGF23 expression in erythropoietic cells of bone marrow thereby contributing to the increase of circulating FGF23 in AKI. We found that erythroid progenitor cells of bone marrow express FGF23. Erythropoietin increased FGF23 expression in vivo and in bone marrow cell cultures via the homodimeric erythropoietin receptor. In experimental AKI secondary to hemorrhagic shock or sepsis in rodents, there was a rapid increase of plasma erythropoietin, and an induction of bone marrow FGF23 expression together with a rapid increase of circulating FGF23. Blockade of the erythropoietin receptor fully prevented the induction of bone marrow FGF23 and partially suppressed the increase of circulating FGF23. Finally, there was an early increase of both circulating FGF23 and erythropoietin in a cohort of patients with severe sepsis who developed AKI within 48 hours of admission. Thus, increases in plasma erythropoietin and erythropoietin receptor activation are mechanisms implicated in the increase of plasma FGF23 in AKI.

Palabras clave

Palabras clave de autor: [acute kidney injury](#); [bone](#); [erythropoietin](#); [FGF23](#); [sepsis](#)

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Información del autor

Dirección para petición de copias: Michea, L (autor para petición de copias)

+ Univ Chile, Fac Med, 1027 Independencia Ave, Santiago 8380453, RM, Chile.

Direcciones:

- + [1] Univ Chile, Inst Ciencias Biomed, Fac Med, Santiago, Chile
- + [2] Hosp Clin Univ Chile, Div Nephrol, Dept Med, Santiago, Chile
- + [3] Hosp Clin Univ Chile, Ctr Invest Clin Avanzada, Santiago, Chile
- [4] Millennium Inst Immunol & Immunotherapy, Santiago, Chile
- + [5] Univ Andres Bello, Fac Biol Sci, Ctr Biomed Res, Santiago, Chile
- + [6] Univ Andres Bello, Fac Med, Santiago, Chile
- + [7] Univ Andes, Fac Med, Dept Obstet & Gynaecol, Santiago, Chile
- + [8] Tokyo Med & Dent Univ, Tokyo, Japan
- + [9] Hosp Clin Univ Chile, Clin Lab, Santiago, Chile
- [10] Millennium Nucleus Ion Channel Associated Dis MiN, Santiago, Chile
- + [11] Hosp Clin Univ Chile, Dept Med, Crit Care Unit, Santiago, Chile

Direcciones de correo electrónico: lmichea@med.uchile.cl

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ELSEVIER SCIENCE INC, 360 PARK AVE SOUTH, NEW YORK, NY 10010-1710 USA

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