

CORRECTION

Correction: mPGES-1 null mice are resistant to bleomycin-induced skin fibrosis

Matthew R McCann^{1†}, Roxana Monemdjou^{2†}, Parisa Ghassemi-Kakroodi¹, Hassan Fahmi¹, Gemma Perez², Shangxi Liu¹, Xu Shi-wen³, Sunil K Parapuram¹, Fumiaki Kojima⁴, Christopher P Denton³, David J Abraham³, Johanne Martel-Pelletier², Leslie J Crofford⁴, Andrew Leask^{1†} and Mohit Kapoor^{2*†}

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Correction

Following publication of our recent article [1], an error in Figure 5d was noticed. In Figure 5d, the β-actin blot corresponding to α-SMA blot was captured on the chemiluminescence imaging system in an inverted orientation. Since this imager does not capture the protein marker, an error in the orientation of the membrane while capturing the picture of the blot occurred. During preparation of the figures for Figure 5d, the α-SMA blot was in right orientation but since orientation of β-actin blot was inverse as it was captured inversely resulted in non-corresponding β -actin blot with respect to α -SMA blot. We have rectified this error now and β -actin blot now corresponds to α-SMA blot. The corrected figure 5 is given here as Figure 1 and a supplementary figure 1 (Additional file 1) showing another set of blots representing Figure 5d is also provided.

¹The Canadian Institute of Health Research Group in Skeletal Development and Remodeling, Division of Oral Biology and Department of Physiology and Pharmacology, Schulich School of Medicine and Dentistry, University of Western Ontario, Dental Sciences Building, London, Ontario, N6A 5C1, Canada. ²Osteoarthritis Research Unit, University of Montreal Hospital Research Center (CR-CHUM) and Department of Medicine, University of Montreal, 1560 Rue Sherbrooke Est, Montréal, Québec, H2L 4M1, Canada. ³Centre for Rheumatology, Department of Medicine, University College London (Royal Free Campus), Rowland Hill Street, London, NW3 2PF, UK. 4Division of Rheumatology, Department of Internal Medicine, University of Kentucky, 740 S. Limestone Street, J-509 Kentucky Clinic, Lexington, KY 40536, USA

Additional material

Additional file 1

Supplementary Figure 1. mPGES-1 genetic deletion results in reduced a-SMA expression in response to bleomycin treatment.

Description. Protein extracts from skin tissue after 4 weeks of bleomycin or PBS treatment were subjected to Western blot analysis with an anti- α -SMA antibody. mPGES-1 null mice treated with bleomycin showed reduced a-SMA expression compared with bleomycin-treated WT mice. Representative blot from four separate animals/group/genotype is shown. (b) Graph represents α-SMA expression normalized to β -actin expression from four separate animals/group/genotype. *P < 0.05; bleomycin-treated WT and mPGES-1 null mice compared with PBS-treated mice. ^+P < 0.05; bleomycin-treated mPGES-1 null mice compared with bleomycintreated WT mice. α-SMA, alpha-smooth muscle actin; β-actin, betaactin; mPGES-1, microsomal prostaglandin E₂ synthase-1.

Format. JPG. Download file at: http://arthritis-research.com/content/ supplementary/ar3285-s1.jpg

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References

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²Osteoarthritis Research Unit, University of Montreal Hospital Research Center (CR-CHUM) and Department of Medicine, University of Montreal, 1560 Rue Sherbrooke Est, Montréal, Québec, H2L 4M1, Canada Full list of author information is available at the end of the article



^{*}Correspondence: mohit.kapoor.chum@ssss.gouv.qc.ca †Contributed equally

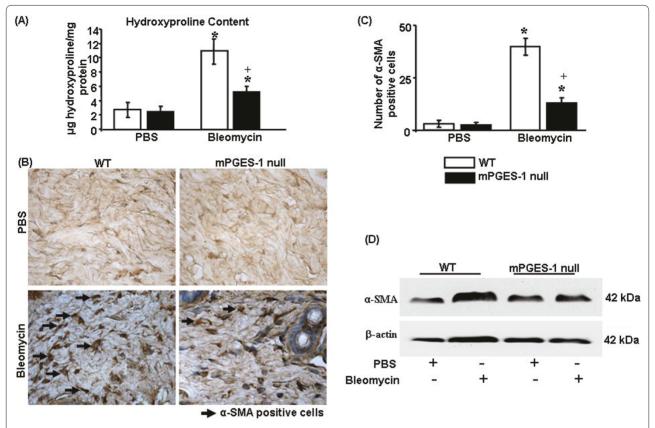


Figure 1. mPGES-1 genetic deletion results in reduced collagen content and myofibroblast formation *in vivo*. (a) Hydroxyproline analysis showed reduced collagen content in mPGES-1 null mice compared with wild-type (WT) mice in response to bleomycin treatment. Data from four separate mice per group are shown. (b, c) Immunohistochemistry using anti-α-SMA antibody was performed. mPGES-1 null mice showed a reduced number of α-SMA-expressing myofibroblasts compared with WT mice in response to bleomycin treatment (4-week treatment). Representative data from four separate animals per group are shown. *P < 0.05; bleomycin-treated WT and mPGES-1 null mice compared with phosphate-buffered saline (PBS)-treated mice. *P < 0.05; bleomycin-treated mPGES-1 null mice compared with bleomycin-treated WT mice. (d) Protein extracts from skin tissue after 4 weeks of bleomycin or PBS treatment were subjected to Western blot analysis with an anti-α-SMA antibody. mPGES-1 null mice treated with bleomycin showed reduced α-SMA expression compared with bleomycin-treated WT mice. Representative blot from four separate animals per group is shown. α-SMA, alpha-smooth muscle actin; mPGES-1, microsomal prostaglandin E₂ synthase-1.