Author's response to reviews

Title: Mitochondria-targeted antioxidant MitoQ ameliorates experimental mouse colitis by suppressing NLRP3 inflammasome-mediated inflammatory cytokines

Authors:

Amarjargal Dashdorj (amka0206@gmail.com)
Jyothi K.R. (jyothibiosci@gmail.com)
Sangbin Lim (dark2lsb@nate.com)
Ara Jo (ahahyeah@naver.com)
Nam Minh Nguyen (minhnam1984@gmail.com)
Joohan Ha (hajh@khu.ac.kr)
Kyung-Sik Yoon (sky9999@khu.ac.kr)
Hyo Jong Kim (hjkim@khmc.or.kr)
Jae-Hoon Park (jhpark@khu.ac.kr)
Michael P Murphy (mpm@mrc-mbu.cam.ac.uk)
Sung Soo Kim (sgskim@khu.ac.kr)

Version: 3 Date: 10 April 2013

Author's response to reviews: see over
Dear Dr. Sabina Alam,

We are happy to submit our manuscript, “Mitochondria-targeted antioxidant MitoQ ameliorates experimental mouse colitis by suppressing NLRP3 inflammasome-mediated inflammatory cytokines” for consideration of possible publication in the research articles of the BMC Medicine. This paper has not been published or accepted for publication and it is not under consideration for publication elsewhere.

Regarding reviewers, we recommend the following experts as our nominees:

1. Holger K. Eltzschig, M.D., Ph.D.
   Department of Anesthesiology
   University of Colorado
   Building 500 - 13001 E. 17th Place,
   Campus Box C290, Aurora, CO 80045.
   Phone: 303-724-2934
   Fax: 720-848-7375
   Email: holger.eltzschig@ucdenver.edu

2. Kevin Moore M.D., Ph.D.
   University College London Medical School
   Royal Free Campus, Centre for Hepatology
   Rowland Hill Street,
   London NW3 2PF, UK
   Phone: 0207 433 2876
   Email: kevin.moore@ucl.ac.uk
Many investigators have demonstrated the correlation between oxidative stress and IBD. However, targeting mitochondrial ROS has not been investigated until now.

Our results clearly showed that ROS originated form mitochondria play a causative role in the pathogenesis of IBD. ROS levels, oxidative injury and inflammatory cytokines IL-1β and IL-18 were increased in colon tissue of DSS-induced colitis mice and significantly decreased by treating with MitoQ. Also, excessive activation of NLRP3 inflammasome which is responsible for those cytokines was suppressed by MitoQ. Taken together, we conclude that mtROS play a critical role in the pathogenesis of IBD and MitoQ is a possible therapeutic molecule for the treatment of IBD.

We believe that our current study is valuable and that our findings will be interesting to many audiences of the BMC-Medicine. Also, our results seem to fit in the scope of the BMC-Medicine.

We hope that our manuscript is considered significant enough for acceptance BMC-Medicine. We would greatly appreciate your reviewing of our manuscript and are looking forward to hearing good news from you soon.

Thank you very much.

Sincerely,

[Signature]