Author's response to reviews

Title: Soluble receptor for advanced glycation end products as an indicator of pulmonary vascular injury after cardiac surgery

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Author's response to reviews: see over
Dear Editor,

Please find enclosed our revised manuscript entitled “Soluble receptor for advanced glycation end products as an indicator of pulmonary vascular injury after cardiac surgery”.

We revised our manuscript accordingly, highlighting all changes in the revised manuscript. A point–by–point reply to all comments is provided in an additional file.

We look forward to your response.

Pieter Roel Tuinman, corresponding author
Reviewer 1:

Minor Essential Revisions

The authors have responded and this is a much improved manuscript. Now, there is one last problem they have to clarify. As I have said that “they could not make the conclusions that the lung vascular injury after cardiac surgery was mediated by sRAGE”. In my opinion, the conclusions of an article were the summary of results, but not inference. According to their findings, they just explored the relationship of plasma sRAGE levels and the development of lung vascular injury after cardiac surgery. In addition, in the logistic regression, the operation time was also independent associated with the occurrence of lung injury. In fact, the elevated levels of sRAGE were correlated with the operation time and cardiopulmonary bypass time according to previous studies. The operation make the sRAGE level elevate and the pulmonary leak index increase in the present study, presumably. So they can only conclude that sRAGE is a biomarker, but not a mediator of lung vascular injury.

*We changed according to the suggestion of the reviewer the conclusion of the abstract: “sRAGE is elevated in plasma after cardiac surgery and indicates increased pulmonary vascular permeability. The level of sRAGE is not affected by transfusion.” (instead of: suggest a mediating role etc.)*

*Furthermore we changed the first paragraph of the discussion as follows: “Although sRAGE levels are increased after cardiac surgery, a mediating role of endogenous AGES in alveocapillary inflammation cannot be concluded from our results altogether.”*