Reply to referee Berkant Muammer Kayatekin:

1. In the new version of the manuscript we improved the description and details of the experimental design. As suggested, we used numbers to the different groups of treatments and specified the numbers of animals used in each treatment. This information is now included in the "Methods" section. Additionally, figures and legends are also re-designed.
2. The order of the sections of the manuscript was changed and page numbers included.
3. Ethical approval was included in the "Material and Methods" section ("Animals" sub-section).
4. We have detailed the description, and added the reference of the electrical stimulation protocol in the "Methods" section. We also added in the second paragraph of the "Results" section: "We have chosen an exercise protocol that involved a polymetric (eccentric) muscle component. Untrained exercised rats therefore experience both oxidative and polymetric stress, with the latter causing some muscle trauma, associated with over-exertion, leading to inflammation."
5. In Figure 2 we have not checked the inflammatory process at "0" (immediately after exercise) or at 1 h after exercise because we thought it would be a very short period to see a significant increase in macrophage density in relation to the control. We also measured macrophage infiltration 48 h after exercise in order to check its effectiveness against the delayed-onset of muscle trauma.
6. Regarding the comments about Fig. 3, the other time-points (brain at 6 h and liver at 48 h after exercise) were not checked because we choose only to analyze the times after exercise where the highest level of lipid peroxidation was detected. In the case of TA muscle, the time-points of 6 h and 48 h after exercise were checked because in these periods lipid peroxidation was still very increased, when compared to the control ones (treatment 1). This information is given in the "Results" section, and showed in Fig. 3A.
7. The reference of Kayatekin et al. (2002) was corrected.

We thank very much for your suggestions, which certainly collaborated to improve the final version of the manuscript.

Yours Sincerely,
Ione Salgado.

Reply to referee Mark Miller:

1. As suggested we have placed the data in the context of trauma associated with over-exertion, leading to inflammation.
2. The order of the sections of the manuscript was changed and page numbers included.
3. As suggested, we have now discussed that nitrination reactions can also be the result of nitrite and myeloperoxidase, as shown in page 15, paragraph 1 of "Discussion" section.
4. We have corrected the description of the CK activity measurements. The highest CK activity was observed already 1 h after exercise and remained up at 6 h after exercise.
5. The cell damage resulted from acute exercise is now discussed (second paragraph in the "Discussion" section).
6. Oral administration of the HCE alone did not affect the density of macrophages in TA muscle. As can be seen in Fig. 2, there is no difference in macrophage cell number between controls (treatment 1) and HCE treated samples (treatment 3).
7. The figure legends were improved, as suggested.
8. Thanks for the suggestion of the histological analysis of protein nitration. Although this is not the scope of the present work, it will be considered for future research.

9. As requested we discussed the oxidative events in brain and liver and related it to trauma caused by acute exercise, as can be seen in the "Discussion" section, page 14, first paragraph.

We thank very much for your suggestions, which certainly collaborated to improve the final version of the manuscript.

Yours Sincerely,
Ione Salgado.