



Firzan Nainu <firzannainu@unhas.ac.id>

BBADIS-23-269: Dr. Paul Murphy invites you to review for BBA - Molecular Basis of Disease

1 message

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To: Firzan Nainu <firzannainu@unhas.ac.id>

Sun, Apr 2, 2023 at 12:57 PM

Manuscript No.: BBADIS-23-269

Title: Functional characterisation of the ACE2 orthologues in *Drosophila* provides insights into the neuromuscular complications of COVID-19

Article Type: Regular Paper

Journal Title: BBA - Molecular Basis of Disease

Corresponding Author: Prof. Ruben J Cauchi

All Authors: Paul Herrera; Ruben J Cauchi

Submit Date: Mar 14, 2023

Dear Dr. Nainu:

I would be grateful if you would review a paper entitled "Functional characterisation of the ACE2 orthologues in *Drosophila* provides insights into the neuromuscular complications of COVID-19" for the journal BBA - Molecular Basis of Disease.

The manuscript abstract appears at the end of this message.

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The manuscript reference number is BBADIS-23-269.

If you agree to review, if possible, the journal would very much appreciate receiving your report within 12 days. An email will be sent to you with reviewer instructions. You may submit your comments online at the above URL. There you will find spaces for confidential Comments to the Editor (including a reviewer form to be completed) and Comments for the Author.

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Sincerely,

Paul Murphy, PhD
Executive Editor
BBA - Molecular Basis of Disease

ABSTRACT:

SARS-CoV-2, the virus responsible for the coronavirus disease of 2019 (COVID-19), gains cellular entry via interaction with the angiotensin-converting enzyme 2 (ACE2) receptor of host cells. Although SARS-CoV-2 mainly targets the respiratory system, the neuromuscular system also appears to be affected in a large percentage of patients with acute or chronic COVID-19. The cause of the well-described neuromuscular manifestations resulting from SARS-CoV-2 infection remains unresolved. These may result from the neuromuscular-invasive capacity of the virus leading to direct injury. Alternatively, they may be the consequence of ACE2 inactivation either due to viral infection, ACE2 autoantibodies or both. Here, we made use of the *Drosophila* model to investigate whether ACE2 downregulation is sufficient to induce neuromuscular phenotypes. We show that moderate gene silencing of ACE2 orthologues *Ance* or *Ance3* diminished survival on exposure to thermal stress, the magnitude of which was increased on induction of neuromuscular fatigue driven by increased physical activity. A strong knockdown of *Ance* or *Ance3* directed to muscle reduced or abolished adult viability and caused obvious motoric deficits including reduced locomotion and impaired flight capacity. Selective knockdown of *Ance* and *Ance3* in neurons caused wing defects and an age-dependent decline in motor behaviour, respectively, in adult flies. Interestingly, RNA sequencing allowed us to discover several differentially spliced genes that are required for synaptic function downstream of *Ance* or *Ance3* depletion. Our findings are therefore supportive of the notion that loss of a RAS-independent function for ACE2 contributes to the neuromuscular manifestations associated with SARS-CoV-2 infection.

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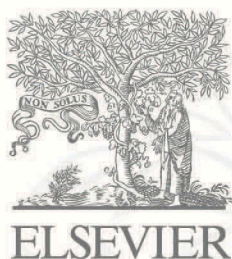
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Biochimica et Biophysica Acta:
Molecular Basis of Disease

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FIRZAN NAINU

in recognition of the review contributed to the journal

The Editors of Biochimica et Biophysica Acta: Molecular Basis of Disease

