

Neurodegeneration in human brain organoids infected with herpes simplex virus type 1

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The Manuscript Microscope Sentence Audit is a research paper introspection system that parses the text of your manuscript into minimal sentence components for faster, more accurate, enhanced proofreading.

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- **Accelerated Proofreading:** Examine long technical texts in a fraction of the usual time.
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Manuscript Authors: Agnieszka Rybak-Wolf, Emanuel Wyler, Ivano Legnini, Anna Loewa, Petar Glažar, Seung Joon Kim, Tancredi Massimo Pentimalli, Anna Oliveras Martinez, Benjamin Beyersdorf, Andrew Woehler, Markus Landthaler & Nikolaus Rajewsky

Features of the Sentence Audit:

The Sentence Audit combines two complementary proofreading approaches:

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The combined approaches ensure easier, faster, more effective proofreading.

Comments and Caveats:

- The sentence parsing is achieved using a prototype natural language processing pipeline written in Python and may include occasional errors in sentence segmentation.
- Depending on the source of the input text, the Sentence Audit may contain occasional html artefacts that are parsed as sentences (E.g. "Download figure. Open in new tab").
- Always consult the original research paper as the true reference source for the text.

Contact Information:

To get a Manuscript Microscope Sentence Audit of any other research paper, simply forward any copy of the text to John.James@OxfordResearchServices.com.

All queries, feedback or suggestions are also very welcome.

Research Paper Sections:

The sections of the research paper input text parsed in this audit.

[illegible]

Title **Neurodegeneration in human brain organoids infected with herpes simplex virus type 1**

S1 [001] Abstract

S1 [002] Herpes simplex virus type 1 (HSV-1) infection of the nervous system may lead to brain damage, including neurodegeneration.

Herpes simplex virus type 1 ...
... (HSV-1) ...
... infection ...
... of the nervous system may lead ...
... to brain damage, ...
... including neurodegeneration.

S1 [003] However, lack of suitable experimental models hinders understanding molecular mechanisms and cell-type-specific responses triggered by HSV-1.

However, ...
... lack ...
... of suitable experimental models hinders understanding molecular mechanisms ...
... and cell-type-specific responses triggered ...
... by HSV-1.

S1 [004] Here, we infected human brain organoids with HSV-1.

Here, ...
... we infected human brain organoids ...
... with HSV-1.

S1 [005] Known features of HSV-1 infection such as alteration of neuronal electrophysiology and induction of antisense transcription were confirmed.

Known features ...
... of HSV-1 infection ...
... such as alteration ...
... of neuronal electrophysiology ...
... and induction ...
... of antisense transcription were confirmed.

S1 [006] Full-length mRNA-sequencing revealed aberrant 3' end formation and poly(A)-tail lengthening.

Full-length mRNA-sequencing revealed aberrant 3' end formation ...
... and poly(A)-tail lengthening.

S1 [007] Single-cell RNA-seq and spatial transcriptomics uncovered changes in the cellular composition of the infected organoids caused by viral replication and dysregulation of molecular pathways in cell-type specific manner.

Single-cell RNA-seq ...
... and spatial transcriptomics uncovered changes ...
... in the cellular composition ...
... of the infected organoids caused ...
... by viral replication ...
... and dysregulation ...
... of molecular pathways ...
... in cell-type specific manner.

S1 [008] Furthermore, hallmarks of early neurodegeneration were observed, namely extracellular matrix disruption, STMN2 and TARDBP/TDP43 downregulation, and upregulation of the AD-related non-coding RNA BC200/BCYRN1.

Furthermore, ...
... hallmarks ...
... of early neurodegeneration were observed, ...
... namely extracellular matrix disruption, ...
... STMN2 ...
... and TARDBP/TDP43 downregulation, ...
... and upregulation ...
... of the AD-related non-coding RNA BC200/BCYRN1.

S1 [009] These hallmarks were weaker/absent when infecting with a mutant HSV-1 control.

These hallmarks were weaker/absent ...
... when infecting ...
... with a mutant HSV-1 control.

S1 [010] Together, our data indicate that brain organoids serve as a powerful model to study mechanisms of HSV-1-driven neurodegeneration.

Together, ...
... our data indicate ...
... that brain organoids serve ...
... as a powerful model ...
... to study mechanisms ...
... of HSV-1-driven neurodegeneration.

S2 [011] Introduction

S2 [012] Herpes simplex virus type 1 (HSV-1) is a common human-specific pathogen affecting a large part of the population worldwide (1).

Herpes simplex virus type 1 ...
... (HSV-1) ...
... is a common human-specific pathogen affecting a large part of the population worldwide ...
... (1).

S2 [013] After primary replication in epithelial cells, HSV-1 travels along axons innervating the affected regions to the trigeminal ganglia to establish a latent state of infection.

After primary replication ...
... in epithelial cells, ...
... HSV-1 travels ...
... along axons innervating the affected regions ...
... to the trigeminal ganglia ...
... to establish a latent state ...
... of infection.

S2 [014] The latent HSV-1 genome persists in episomal form within the nucleus, and HSV-1 DNA is chromatinized with heterochromatic histone marks.

The latent HSV-1 genome persists ...
... in episomal form ...
... within the nucleus, ...
... and HSV-1 DNA is chromatinized ...
... with heterochromatic histone marks.

S2 [015] In this state, only a small subset of viral genes is transcribed, including the latency-associated transcripts (LATs) (2, 3).

In this state, ...
... only a small subset ...
... of viral genes is transcribed, ...
... including the latency-associated transcripts ...
... (LATs) ...
... (2, 3) ...
... .

S2 [016] Investigations of post-mortem human tissue have provided evidence of LAT transcription/virus not only in trigeminal ganglia (4) but also in the brain at large (3).

Investigations ...
... of post-mortem human tissue have provided evidence ...
... of LAT transcription/virus not ...
... only ...
... in trigeminal ganglia ...
... (4) ...
... but also ...
... in the brain ...
... at large ...
... (3).

S2 [017] Latent viral infections can persist over years with very limited transcription.

Latent viral infections can persist ...
... over years ...
... with very limited transcription.

S2 [018] However, stimuli such as stress signals and weakened immunity can cause the reactivation of HSV-1 from sensory neurons at any time (3).

However, ...
... stimuli ...
... such as stress signals ...
... and weakened immunity can cause the reactivation ...
... of HSV-1 ...
... from sensory neurons ...
... at any time ...
... (3).

S2 [019] Two different forms of infection-related diseases in the brain have been observed.

Two different forms ...
... of infection-related diseases ...
... in the brain have been observed.

S2 [020] First, lytic infection, either by reactivation from latency or primary infection (5) in the central nervous system (CNS) can cause herpes simplex encephalitis (HSE), with an incidence of 1 in 10,000 infected individuals (6).

First, ...
... lytic infection, ...
... either ...
... by reactivation ...
... from latency ...
... or primary infection ...
... (5) ...
... in the central nervous system ...
... (CNS) ...
... can cause herpes simplex encephalitis ...
... (HSE), ...
... with an incidence ...
... of 1 ...
... in 10,000 infected individuals ...
... (6).

S2 [021] HSV-1 infection in CNS is the most common cause of viral encephalitis (7).

HSV-1 infection ...
... in CNS is the most common cause ...
... of viral encephalitis ...
... (7).

S2 [022] Second, HSV-1 is gaining increasing attention as a potentially causative agent in the pathogenesis of sporadic Alzheimer's disease (AD) (8–12).

Second, ...
... HSV-1 is gaining increasing attention ...
... as a potentially causative agent ...
... in the pathogenesis ...
... of sporadic Alzheimer's disease ...
... (AD) ...

End of Sample Audit

This is a truncated Manuscript Microscope Sample Audit.

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