A Manuscript Microscope 'Sentence Audit' of:

Pathogenic tau disrupts the cellular program that maintains neuronal identity

What is the Manuscript Microscope Sentence Audit?

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.

Why use a Sentence Audit to proofread your manuscript?

- Accelerated Proofreading: Examine long technical texts in a fraction of the usual time.
- Superior Proofreading: Detect subtle errors that are invisible to traditional methods.
- Focused Proofreading: Inspect each individual sentence component in isolation.
- Reliable Proofreading: Ensure every single word of your manuscript is correct.
- Easier Proofreading: Take the hardship out of crafting academic papers.

Bonus 1: Improved Productivity: Rapidly refine rough drafts to polished papers.
Bonus 2: Improved Authorship: Cultivate a clear, concise, consistent, writing style.
Bonus 3: Improved Reputation: Become known for rigorously precise publications.

Manuscript Source: https://www.biorxiv.org/content/10.1101/2021.03.05.434166v1

Manuscript Authors: Adrian Beckmann, Paulino Ramirez, Maria Gamez, William J. Ray & Bess Frost

Audit Date: 22/03/21 **Audit Identifier:** 2579ZK7376K5020 **Code Version:** 3.6

Features of the Sentence Audit:

The Sentence Audit combines two complementary proofreading approaches:

1. Each sentence of your text is parsed and displayed in isolation for focused inspection.
2. Each individual sentence is further parsed into Minimal Sentence Components for a deeper review of the clarity, composition and consistency of the language you used.

The Minimal Sentence Components shown are the smallest coherent elements of each sentence of your text as derived from it's conjunctions, prepositions and selected punctuation symbols (i.e. commas, semicolons, round and square brackets).

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.

Section No.	Headings	Sentences
Section: 1	Abstract	8
Section: 2	Introduction	16
N/A	--- --- --- --- --- --- ---	0
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The Sentence Audit Of The Research Paper

Title **Pathogenic tau disrupts the cellular program that maintains neuronal identity**

S1 [001] Abstract

S1 [002] Neurons in human Alzheimer’s disease acquire phenotypes that are also present in various cancers, including over-stabilization of the cytoskeleton, nuclear pleomorphism, decondensation of constitutive heterochromatin, and aberrant activation of the cell cycle.

Neurons ...
... in human Alzheimer’s disease acquire phenotypes ...
... that are also present ...
... in various cancers, ...
... including over-stabilization ...
... of the cytoskeleton, ...
... nuclear pleomorphism, ...
... decondensation ...
... of constitutive heterochromatin, ...
... and aberrant activation ...
... of the cell cycle.

S1 [003] Unlike in cancer, in which cell cycle activation drives tumor formation, activation of the cell cycle in post-mitotic neurons is sufficient to induce neuronal death.

Unlike ...
... in cancer, ...
... in which cell cycle activation drives tumor formation, ...
... activation ...
... of the cell cycle ...
... in post-mitotic neurons is sufficient ...
... to induce neuronal death.

S1 [004] Multiple lines of evidence suggest that abortive cell cycle activation is a consequence of pathogenic forms of tau, a protein that drives neurodegeneration in Alzheimer’s disease and related “tauopathies.”

Multiple lines ...
... of evidence suggest ...
... that abortive cell cycle activation is a consequence ...
... of pathogenic forms ...
... of tau, ...
... a protein ...
... that drives neurodegeneration ...
... in Alzheimer’s disease ...
... and related “tauopathies.” ...
...

S1 [005] We have combined network analysis of human Alzheimer's disease and mouse tauopathy with mechanistic studies in Drosophila to discover that pathogenic forms of tau drive abortive cell cycle activation by disrupting the cellular program that maintains neuronal identity.

We have combined network analysis ...
... of human Alzheimer's disease ...
... and mouse tauopathy ...
... with mechanistic studies ...
... in Drosophila ...
... to discover ...
... that pathogenic forms ...
... of tau drive abortive cell cycle activation ...
... by disrupting the cellular program ...
... that maintains neuronal identity.

S1 [006] Mechanistically, we identify Moesin, a prognostic biomarker for cancer and mediator of the epithelial-mesenchymal transition (EMT), as a major effector of tau-induced neurotoxicity.

Mechanistically, ...
... we identify Moesin, ...
... a prognostic biomarker ...
... for cancer ...
... and mediator ...
... of the epithelial-mesenchymal transition ...
... (EMT), ...
... as a major effector ...
... of tau-induced neurotoxicity.

S1 [007] We find that aberrant activation of Moesin in neurons acts through the actin cytoskeleton to dysregulate the cellular program that maintains neuronal identity.

We find ...
... that aberrant activation ...
... of Moesin ...
... in neurons acts ...
... through the actin cytoskeleton ...
... to dysregulate the cellular program ...
... that maintains neuronal identity.

S1 [008] Our study identifies mechanistic parallels between tauopathy and cancer and sets the stage for novel therapeutic approaches.

Our study identifies mechanistic parallels ...
... between tauopathy ...
... and cancer ...
... and sets the stage ...
... for novel therapeutic approaches.

S2 [009] Introduction

S2 [010] Post-mitotic cells such as neurons require persistently active cellular controls to maintain a quiescent, non-cycling, state of terminal differentiation1–3.

Post-mitotic cells ...
... such as neurons require persistently active cellular controls ...
... to maintain a quiescent, ...
... non-cycling, ...
... state ...
... of terminal differentiation1–3.

S2 [011] A curious aspect of postmortem human Alzheimer's disease brains as well as brains of multiple animal models of Alzheimer's disease and related tauopathies is the neuronal upregulation of proteins that are associated with cell cycle activation4,5.

A curious aspect ...
... of postmortem human Alzheimer's disease brains ...
... as well ...
... as brains ...
... of multiple animal models ...
... of Alzheimer's disease ...
... and related tauopathies is the neuronal upregulation ...
... of proteins ...
... that are associated ...
... with cell cycle activation4,5.

S2 [012] Unlike cancer, in which uncontrolled cell division causes tumor formation, cell cycle activation in post-mitotic neurons causes neuronal death rather than neuronal division6–9.

Unlike cancer, ...
... in which uncontrolled cell division causes tumor formation, ...
... cell cycle activation ...
... in post-mitotic neurons causes neuronal death rather than neuronal division6–9.

S2 [013] Cellular phenotypes beyond cell cycle activation are shared between tauopathy and various cancers, including over-stabilization of the actin cytoskeleton10–13, changes in nuclear shape and the lamin nucleoskeleton14,15 and loss of heterochromatin-mediated transcriptional silencing16,17 all of which are also known to be important determinants of cellular identity18–20.

Cellular phenotypes ...
... beyond cell cycle activation are shared ...
... between tauopathy ...
... and various cancers, ...
... including over-stabilization ...
... of the actin cytoskeleton10–13, ...
... changes ...
... in nuclear shape ...
... and the lamin nucleoskeleton14,15 ...
... and loss ...
... of heterochromatin-mediated transcriptional silencing16,17 all of ...
... which are also known ...
... to be important determinants ...
... of cellular identity18–20.

S2 [014] Some basic biological functions require dynamic shifts between programs that control cellular identity and those that promote cellular plasticity.

Some basic biological functions require dynamic shifts ...
... between programs ...
... that control cellular identity ...
... and those ...
... that promote cellular plasticity.

S2 [015] During EMT, for example, transdifferentiation of epithelial cells into mesenchymal cells is important for wound healing21,22 and organ development23.

During EMT, ...
... for example, ...
... transdifferentiation ...
... of epithelial cells ...
... into mesenchymal cells is important ...
... for wound healing21,22 ...
... and organ development23.

S2 [016] Mechanistically, the cytoskeletal remodeling that occurs with EMT causes breakdown of cell-to-cell connections and depletion of proteins that maintain a terminally differentiated epithelial identity.

Mechanistically, ...
... the cytoskeletal remodeling ...
... that occurs ...
... with EMT causes breakdown ...
... of cell-to-cell connections ...
... and depletion ...
... of proteins ...
... that maintain a terminally differentiated epithelial identity.

S2 [017] A shift from cellular identity to cellular plasticity can also mediate disease.

A shift ...
... from cellular identity ...
... to cellular plasticity can also mediate disease.

S2 [018] In cancer, for example, EMT disrupts the terminally differentiated epithelial phenotype to facilitate tumor metastasis24,25, cell cycle activation and consequent malignancy26,27,28.

In cancer, ...
... for example, ...
... EMT disrupts the terminally differentiated epithelial phenotype ...
... to facilitate tumor metastasis24,25, ...
... cell cycle activation ...
... and consequent malignancy26,27,28.

S2 [019] In mature neurons, a terminally differentiated state is maintained by “terminal neuronal selector proteins,” key transcription factors that are in part regulated by the extracellular environment29–32.

End of Sample Audit

This is a truncated Manuscript Microscope Sample Audit.

To get the full audit of this text (or any other research paper),
forward a copy of the research paper to John James at
John.James@OxfordResearchServices.com
